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CUTANEOUS AND INTRACUTANEOUS TUBERCULIN TESTS IN INFANTS AND CHILDREN*

MURRAY H. BASS, M.D.
NEW YORK

With the introduction of simple and harmless methods for the determination of the presence or absence of tuberculous infection, our knowledge of the prevalence of tuberculosis has made immense strides. The work of von Pirquet, Schick, Hamburger, Ghon and many others has taught us that tuberculous infection is common, much more common than we had previously believed; that its incidence in childhood increases as the age of the child advances; that tuberculous infection is by no means synonymous with tuberculous disease. Moreover, we are now in possession of much evidence which proves (1) that the respiratory tract is in a very large percentage of cases the portal of entry; (2) that the bronchial lymph nodes are the seat of tubercles in almost all children who react positively to tuberculin tests; and (3) that the great majority of cases of adult pulmonary tuberculosis probably depend on these very nodes for their source of origin.

In spite of all this advance in our knowledge, however, there is still a great deal to be learned about the tuberculin reaction as it affects children.[1] We were led to collect some data on this subject by the following occurrence: In our institution, a large asylum for infants and young children, it was customary to perform von Pirquet tests on all the children admitted. Wishing to obtain some information concerning the reaction, we chose a number of children who had reacted positively six months previously, and retested them. To our surprise, quite a number of them failed to react. In many of these children the test was repeated several times in the course of a few days, and the reaction then changed from negative to positive.

This occurrence showed quite definitely that the reactivity of the individual undergoes change, and that although a single positive reaction means the presence of tubercle, the power of response to tuberculin may be so altered by time that the same individual, though undoubtedly still the subject of tuberculous infection, fails to respond to the same tuberculin dose. For all clinical purposes, a single or at

* Submitted for publication Feb. 27, 1918.

* From the Home for Hebrew Infants.

1. An excellent compilation of the literature on this subject may be found in "La Tuberculose de l'Enfant" by L. Jeanneret, Paris, 1915.

most two tests may be sufficient, but when we are attempting to gather statistics as to the frequency of tubercle infection, this is by no means sufficient. In a search of the literature we could find only very few records where systematic attempts had been made, in a large group of children, to push to its limit the possibility of obtaining a positive reaction. We therefore set ourselves the task of repeatedly testing a large number of infants and young children in the hope of uncovering, as it were, many hidden cases of latent tuberculous infection.

Of the various local tuberculin tests devised, the most commonly employed in children have been (1) the von Pirquet test, the cutaneous application of tuberculin through an abrasion in the skin; (2) the *Stichreaction* of Escherich, local redness at the site of a subcutaneous injection of tuberculin; (3) the conjunctival reaction of Wolff-Eisner and Calmette; (4) the cutaneous reaction of Moro, the reaction to tuberculin rubbed into the unabraided skin; (5) the intracutaneous test of Mantoux.

Of these the Moro test and the conjunctival reaction have been generally abandoned. The von Pirquet test, owing to its simplicity, has found the greatest favor, and has been very extensively employed. The *Stichreaction* has been studied by Hamburger, who was able to show that it was much more delicate than the von Pirquet test. The method had been described many years ago by Escherich and Ebstein, but neither had thought it of a specific nature, and it was only after the specificity of the cutaneous test had been shown by von Pirquet and Schick that attention was again drawn to the *Stichreaction*. The positive reaction is described as an area from 18 to 30 mm. wide at the site of the subcutaneous injection, red in color, sometimes raised and often painful. It usually appears from eighteen to twenty-four hours after the injection, increases during the second day, diminishes on the third, and after four or five days leaves a brownish discoloration. Very small amounts of tuberculin are sufficient to give a positive reaction, 0.001 mg. being sufficient. The great advantage of the *Stichreaction* over the cutaneous are twofold: first, it is much more delicate; second, we know the exact dosage and concentration of the tuberculin used, and can therefore, as it were, titrate the patient's susceptibility. Cases which react to 0.001 mg. or more intradermally, usually also react positively to the cutaneous test. On the other hand, those persons with an intradermal test positive to smaller amounts of tuberculin, usually fail to show a positive von Pirquet reaction. According to Hamburger, the majority of active cases have a susceptibility to at least 0.001 mg. Inactive tuberculosis, on the other hand, reacts positively to the cutaneous test in only two thirds to three quarters of the cases.

A modification of this test, for which even greater delicacy is claimed, was introduced by Mantoux and Roux in 1909. Instead of the subcutaneous, they employed the intracutaneous route. They used a 1:5,000 solution of tuberculin in normal salt solution and injected one drop, that is, 0.01 mg., of tuberculin directly into the skin by means

of a very fine needle. They claimed that the positive reaction was well marked, appearing as an infiltration surrounded by a red areola, the reaction attaining its maximum at forty-eight hours.

Subsequent investigators have modified the dosage in this test, some using graduated amounts of tuberculin, thus obtaining information not only as to the absolute presence or absence of tuberculous infection, but also as to the titer of the patient's ability to react.

Since so many reports were encountered in which conclusions were drawn from a single von Pirquet test, and since it seemed definitely proved that the intradermic test was the most delicate, we decided on the following procedure: Each child was given a von Pirquet test which was observed after twenty-four, and again after forty-eight hours. If positive, this was regarded as evidence of tuberculous infection. If negative after forty-eight hours, a second von Pirquet test was performed. After forty-eight hours a third test was made. If this proved negative, an intradermic injection of 0.1 c.c. of 1:1,000 old Koch tuberculin (that is, 0.1 mg.) in saline solution was given. If after forty-eight hours this was negative, 0.1 c.c. of a 1:100 solution (that is, 1 mg.) was injected and repeated after forty-eight hours if negative. If after these six tests the case still failed to react, it was considered definitely negative. In all, 1,165 tests were made on 206 children.[2]

The children tested were inmates of an infant asylum in which the hygienic conditions are of the best. None of the infants or children at the time of testing showed any clinical manifestations of tuberculosis, nor were any of them tested during the course of an acute illness. The children varied in age from a few days up to 6 years. They were drawn from the poorest class of New York City's Hebrew population.

Table 1 will give a general view of our results. In all, fifty-two children showed a positive reaction to some of the tests, giving a percentage of 25.2; that is to say, one quarter of the population of the asylum had been at some time infected with the tubercle bacillus.

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2. The von Pirquet tests were made with undiluted old tuberculin, obtained from the New York City Board of Health Laboratory, a regular von Pirquet scarifier being used. For the intradermal tests an all glass hypodermic syringe and fine needle were used. Care was taken that the needle was thrust into and not under the skin, this being ascertained by the appearance of a tiny wheal as the solution was being forced between the layers of the skin. All tests were made on the flexor surface of the forearm, after cleansing with alcohol, and were read after a period of forty-eight hours, since it is generally conceded that any traumatic action has by that time disappeared.

As these tests were carried out over a period of two years, it was obviously impossible to make use of the same solution of tuberculin for them all. Many of them, however, were done with the same solution, and in no child was different tuberculin used for the different tests; that is to say, the three von Pirquet and the three intradermic tests were always performed with the same stock tuberculin.

TABLE 1.—REACTION TO MULTIPLE CUTANEOUS AND INTRACUTANEOUS TUBERCULIN TESTS (206 CHILDREN), 52 POSITIVE REACTIONS

Age	First Test (v. Pirquet) Positive	Second Test (v. Pirquet) Positive	Third Test (v. Pirquet) Positive	Fourth Test Intracutaneous 0.1 Mg. Positive	Fifth Test Intracutaneous 1 Mg. Positive	Sixth Test Intracutaneous 1 Mg. Positive	Total Positive	Total Negative
0- 6 mos.	2	0	0	0	2	0	4	47
6-12 mos.	2	0	1	0	2	1	6	32
1-2 yrs.	0	1	1	1	2	0	5	24
2-3 yrs.	1	3	0	2	5	3	14	31
3-4 yrs.	0	1	3	1	1	0	6	7
4-5 yrs.	1	1	1	2	1	0	6	10
5-6 yrs	1	1	1	7	0	1	11	3

TABLE 2.—RESULTS OF CUTANEOUS AND INTRACUTANEOUS TUBERCULIN TESTS ON 206 CHILDREN, SHOWING THE INCREASED NUMBER OF POSITIVE REACTIONS AS THE TESTS ARE REPEATED

	Number of Positive Cases	Percentage
One cutaneous test	7	3.3
Two cutaneous tests	14	6.8
Three cutaneous tests	21	10.1
Three cutaneous and one intracutaneous tests (0.1 mg.)	34	16.5
Three cutaneous and two intracutaneous tests (0.1 mg.; 1 mg.)	47	22.8
Three cutaneous and three intracutaneous tests (0.1 mg.; 1 mg.; 1 mg.)	52	25.2

Table 2 has been compiled to show the great importance of retesting the negative cases. It will be seen that if we had employed but a single von Pirquet test we should have drawn the incorrect conclusion that only 3.3 per cent. of the inmates had been infected with the tubercle bacillus. If we had used two von Pirquet tests and stopped there, the percentage would have been doubled (6.8), and so on, until by the use of the intradermic tests the true percentage of infected cases was found to be 25.2.

In Table 3 we have arranged the cases according to their ages and have calculated the percentage of positives in each age group. It is very interesting to note the step-like increase in the disease incidence with increasing age. Thus out of fifty-one cases under 6 months of age, four reacted positively, a percentage of 7.8. Of children in the second six months 15.8 per cent. were positive; of those from 1 to 2

years old, 17.2 per cent. were positive; of those aged from 2 to 3 years, 31.3 per cent. were positive; of those aged from 3 to 5 years, 42.8 per cent. were positive; and finally of the children aged from 5 to 6 years, 78 per cent. were positive.

A consideration of these results leads to some interesting deductions. An important fact to be emphasized is that any series of examinations designed to ascertain the incidence of tubercle infection must consist of more than a single test in each individual case. In the case of the intradermal application of tuberculin in milligram doses, the change from a negative to a positive reaction may be supposed to be due to the sensitizing influence of the previous dose. In the case of the von Pirquet test, however, authorities seem to agree that not enough tuberculin enters the organism to change its allergy. Why a second or a third test should be positive when the previous ones were negative remains unexplained.

TABLE 3.—Age Distribution of Patients Reacting Positively to Cutaneous or Intracutaneous Test

Age	Number Tested	Number Reacting Positively	Percentage
0-6 mos.	51	4	7.8
6-12 mos.	38	6	15.8
1-2 yrs.	29	5	17.2
2-3 yrs.	45	14	31.1
3-5 yrs.	28	12	42.8
5-6 yrs.	14	11	78.0

The second fact worthy of note is the great frequency with which we have encountered positive reactions. In most of the American statistics the incidence of the disease in young infants is not nearly so great as we have encountered it, and for this reason objection might be raised to our method or to our interpretation of the reactions. Several factors, however, go to show that we are dealing with true reactions and that our reading of the tests are correct. In the first place, the gradual increase in the percentage of positive cases with increased age of the children cannot be mere coincidence. In Table 3, if we look at the percentage column, we see a steady rise from the percentage of incidence in infants under 6 months, to children in their fifth year of life. (We have grouped together the children in their third to fourth year and those in their fourth to fifth years, since we have had so few at each of these ages.) As is well known, the tuberculin sensitiveness does not become manifest before the tenth week of life (Loewenstein), those weeks being the latent period during which the child forms the antibodies necessary for the reaction. As more and more infections

take place owing to a longer period of exposure, it is quite natural that with increased age, the number of positive reactions should grow larger.

The objection might also be raised that we had confused pseudo reactions with positive reactions. To guard against this, several experiments were performed. First, to make sure of the fact that we were dealing with a specific tuberculin reaction and not some nonspecific skin manifestation, we proceeded as follows: In twenty children, some of whom had reacted positively and others negatively, there was injected intradermally, as control, a solution of broth, peptone and glycerin, prepared in the same manner as in the old Koch tuberculin, omitting, however, the addition of tubercle bacilli. This solution, which was kindly prepared for us by Dr. H. Miller of the bacteriological laboratory of the College of Physicians and Surgeons, was diluted in the same manner as the tuberculin; that is, 1 : 1,000 and 1 : 100, and of this 0.1 c.c. was injected intracutaneously. In not a single instance was

TABLE 4.—Results of Intracutaneous Injections and Reinjections with the Same Dose

Case	Date of First Positive Reaction	Result of Test (Size of Areola in Mm.)	Date of Retest	Result of Retest (Size of Areola in Mm.)
H. T.	May 9	Positive (7×7)	June 8	Positive (7×7)
A. H.	March 2	Positive (7×7)	June 8	Positive (25×25)
H. V.	May 2	Positive (10×10)	June 8	Positive (7×7)
G. U.	May 2	Positive (4×4)	June 8	Positive (20×20)

any reaction noted, either in children who had reacted positively or those who had reacted negatively to tuberculin. This result, we believe, definitely disposes of any objection that might be raised on the ground that we are dealing in some of our cases with a nonspecific reaction.

Further, to prove the correctness of the readings, a number of our positive cases were retested some time after the last positive reactions had been obtained, as will be seen in Tables 4 and 5. In Table 4 are tabulated four cases in which 0.1 mg. tuberculin had caused definite but small sized reactions. On account of the relatively small reactions, these children were retested from one to three months later with a single intracutaneous dose of 0.1 mg. All four patients gave definite, positive reactions.

In Table 5 are shown the records of seven cases chosen at random among that group which had failed to react to the three von Pirquet tests and also to the intracutaneous test with 0.1 mg., the reaction only appearing when 1 mg. was used. Thinking that these children's

reactivity might have been altered with time and with the previous administration of tuberculin, we reinjected them at a period of from two to three months later, with 0.1 mg., a dose to which all of them had previously failed to react. As will be seen in the table, all but one patient showed definite marked positive reactions. This one child, Mary G., was then reinjected forty-eight hours later with 1 mg., to which she reacted with positive reaction similar to the one she had shown two and one half months previously.

We have, therefore, eleven cases which were retested and of these every patient without exception continued to react with a positive reaction.

As a matter of interest, an attempt was made to repeat the 1 mg. intradermal tests on infants who had reacted negatively to the six routine tests. These infants, four in number, were reinjected with

TABLE 5.—RESULTS OF INTRACUTANEOUS INJECTIONS AND SUBSEQUENT REINJECTIONS WITH SMALLER DOSE

Case	Date of Negative Reaction (0.1 Mg.)	Date of Positive Reaction (1 Mg.)	Date of Reinjection (0.1 Mg.)	Result of Reinjection (Diameter of Areola in Mm.)	Remarks
P. S.	April 27	April 30	June 8	Positive (12×12)	
I. R.	April 27	April 30	June 8	Positive (30×30)	
S. S.	April 15	April 18	June 8	Positive (10×12)	
C. S.	April 15	April 18	June 8	Positive (10×10)	
M. G.	March 25	March 28	June 8	0	Retested again June 10 with 1 mg.; positive reaction (12×12)
H. B.	March 25	March 28	June 8	Positive (7×7)	
L. H.	March 25	March 28	June 8	Positive (10×10)	

1 mg. every other day for two weeks (seven additional tests), but all of them remained consistently negative.

When we compare our results with those of other investigators we find that our results are in accord with a number of those who have used the intracutaneous method. Many other series, however, especially those in which the von Pirquet test alone was relied on, show much lower percentage of positive reactions. Table 6 shows the results obtained by investigators who have used the von Pirquet and the intracutaneous tests.

The most recently published large series of American cases is that of Veeder and Johnston.[3] Our results and theirs do not agree, though it is perhaps not fair to compare them, since these authors themselves state that their "figures do not represent a special study of the fre-

3. Veeder, B. S., and Johnston, M. R.: AM. JOUR. DIS. CHILD., 1915, **9**, 478.

quency of tuberculin reactions in childhood, but the results of tests made for diagnostic purposes on hospital patients regardless of the cause for their admission." As our figures do represent a specially intensive search for positive reactions, it is natural that they should be higher. Then, too, as many authors, including Veeder and Johnston, have pointed out, the environment and social status of the particular group of children investigated must make a vast difference in the figures obtained. In one particular, however, we must absolutely disagree with the last named authors, and that is on the question of the greater delicacy of the intradermic tests as compared with the von Pirquet. Veeder and Johnston say, "The intradermic tuberculin test

TABLE 6.—FREQUENCY OF POSITIVE TUBERCULIN SKIN REACTIONS IN CHILDHOOD ACCORDING TO VARIOUS INVESTIGATORS *

I. Calmette, Grysez and Letulle (1,226 children examined)	Per Cent. Positive
0–1 yr.	8.7
1–2 yrs.	22.1
2–5 yrs.	53.8
5–15 yrs.	81.4

II. Karl Kaspar (Erlangen)	
0–1 yr.	10.0
1–2 yrs.	31.7
2–4 yrs.	30.0
4–14 yrs.	60.0

Méroz and Khalatoff (Geneva)	Per Cent. Positive
0–6 mos.	0.0
6–12 mos.	6.6
1–2 yrs.	17.6
2–3 yrs.	40.0
3–5 yrs.	51.0
5–7 yrs.	61.2
7–10 yrs.	66.4
10–15 yrs.	78.7

Apteckmant (Paris)	
0–1 yrs.	0.5
1–2 yrs.	16.0
2–5 yrs.	43.0
6–10 yrs.	68.0
11–15 yrs.	78.0

Mantoux and Lemaire (300 children examined)	Per Cent. Positive
1–2 yrs.	16.0
2–4 yrs.	51.0
4–7 yrs.	66.0
7–15 yrs.	82.0

Mantoux (174 children examined)	
0–1 yr.	2.5
1–3 yrs.	29.0
3–7 yrs.	50.0
7–15 yrs.	80.0

Morgenroth	
200 infants	5.0

* Quoted from L. Jeanneret: Rev. méd. de la Suisse. romande, 1913, **33**, 871.

has given but a slight increase in the percentage of positive reactions over the percentage of positive cutaneous reactions." In our method of procedure, however, we found only twenty-one positive reactions when the von Pirquet test alone was employed, whereas of the remaining 185 children, thirty-one reacted to the intracutaneous test.

In all probability, if the intracutaneous method had been used alone, to the exclusion of the von Pirquet test, the results would have been identical as far as concerns the discovery of latent tuberculous infection; but we chose to use both the cutaneous and the intracutaneous application in order to compare the two, and also to show that even if one confines oneself to the von Pirquet method, the tests, if negative, ought to be repeatedly performed.

In the course of this work the fact became evident that certain of the children showed very marked local reactions, though their general condition appeared to be excellent and no clinical signs of disease could be elicited. As many of these children belonged to the "exudative diathesis" group, it seemed possible that since they were so prone to skin

eruptions, their specially violent reaction to tuberculin might be correlated with a particularly susceptible skin.

To help clear up this question a number of the children who had shown the most pronounced tuberculin reactions were subjected to different skin irritants.[4] Mustard paste, ointments containing various percentages of cantharides and solutions of various strengths of phenol were applied to the unbroken skin for a definite period of time. Comparisons were then made between the resulting erythema of the skin with that caused by the same irritants in children who had failed to react to the von Pirquet test. All of these attempts to prove any particular skin sensitiveness in the former group of children resulted negatively. We refrain from publishing these experiments in detail since they uniformly failed to explain any cause for the very large size of some of the reactions.

Much work has been done on this particular phase of the reaction; the marked reaction has been explained as the result, on the one hand, of a severe infection, or, on the other, as a sign of marked resistance. That there is, however, a local dermal factor besides the general reactivity of the patient tested, is shown by the following experiment performed by von Pirquet:[5]

In children who had temporarily lost the reaction to tuberculin on account of an attack of measles, a cutaneous test was performed daily on different parts of the body. The reaction on the dorsum of the foot seemed larger than those on the arms, whereas the skin of the neck gave the smallest reactions. Frehn,[6] on the contrary, found the skin of the chest most sensitive.

A critical survey of the literature, moreover, fails to show any unanimity in opinions on the subject. Von Pirquet[7] himself says:

> The cutaneous tuberculin reaction depends on at least two factors. One of these factors is the tuberculin, and the other, furnished by the organism, can be considered as an antibody, whose origin dates back to a previous infection of the organism with tubercle bacilli. . . . In persons who give the reaction, a marked difference exists between different areas of skin. In skin areas which have been subjected previously to one or repeated applications of tuberculin, the reaction proceeds in a different manner. The most prominent influence of the concentration of the antibody seems to lie in the time in which the reaction appears and reaches its maximum, whereas the absolute height of the curve, signifying the intensity of the reaction, seems to be dependent on the strength of the tuberculin.

4. Similar experiments have been reported by Holmgren, I.: Beitr. zur Klin. d. Tuberk., 1912, No. 25; Rachmilewitsch: Jahrb. f. Kinderh., 1913, No. 77; Entz: Wien. klin. Wchnschr., 1908, No. 12, and others.

5. Von Pirquet: Wien. med. Wchnschr., 1913, **39**, 2518.

6. Frehn: Beitr. z. Klin. d. Tuberk., 1914, No. 32.

7. Von Pirquet: Jour. Pharm. and Exper. Therap., 1909-1910, **1**, 174.

A survey of our experiments here reported reveals that infection by the tubercle bacillus is probably as frequent in America as elsewhere, and that to detect the infected children, frequently repeated tests are necessary. The interesting question arises as to the significance of these great numbers of latent cases. Are the children whose susceptibility is so slight that they react only to the intradermal test to be regarded as better, or less well protected against the disease tuberculosis? Is mild susceptibility synonymous with marked resistance, or only with slight infection? At present these questions cannot be answered and their solution can only be arrived at if series of individuals can be followed for longer periods of time. We are now in possession of knowledge which makes us fairly certain that the tuberculous infection acquired in childhood has a distinct bearing on the disease as it appears in later life, and it will be by following up series of cases such as are here reported, that we may arrive at some definite relationship between tuberculin susceptibility and tuberculous disease.

We wish to express our indebtedness to Dr. Alfred Hess for permission to carry on this work and for much helpful advice; also to Dr. Barenberg, Resident Physician at the Home, without whose assistance and cooperation much of this work would have been impossible.

41 West Eighty-Third Street.

THE HYPERTONIC INFANT, THE CURATIVE ACTION OF ATROPIN ON CERTAIN OF ITS MANIFESTATIONS *

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The Hypertonic Infant is a definite clinical entity. It is characterized by hypertonicity of all the skeletal muscles, as shown by the ability to raise the head and grasp objects even in the early days of life, and by general spasticity. The hollow viscera show increased activity of their smooth muscle fibers. This expresses itself in the form of spasm involving practically every part of the digestive tube, and, depending on the region, presents the symptom of colic, visible peristalsis, vomiting, constipation, or any combination of these. Accompanying these symptoms is marked psychic irritability, expressed by insomnia, general restlessness and crying. The hypertonic infant belongs to the *spasmophilic group* and presents the symptoms of *Vagotonia*.[1]

Its recognition is of importance in this, that while presenting symptoms in the main of disturbances of nutrition, food regulation alone does not correct the fault, whereas treatment by the drug *atropin* is followed by rapid subsidence of symptoms, the results being so prompt and regular as to constitute specific action. The tolerance for milk which is usually low in these infants is at once materially increased, so that a normal amount may be utilized—a condition quite opposed to that existing before the atropin was begun.

In the teaching of infant feeding stress has been laid on two factors: the *food* and the *infant*. The food has been studied from many angles, with the result that there is available an ample and satisfactory variety adaptable to most cases.

The infant, too, has been studied from many angles; but few serious attempts have been made to study the possibility of altering the infant's reaction toward food, when it is found impossible to adapt the food to it, despite the fact that idiosyncrasies have been noted and differentiated. Personal experience during the last few years has convinced me that in this direction lies a field which is full of promise in that ever diminishing group of difficult feeding cases. This paper deals with only one phase of the problem and will confine itself to the

* Submitted for publication March 4, 1918.

1. Eppinger and Hess: Vagotonia, Nervous and Mental Diseases, Monograph 20. Ed. 2, Nervous and Mental Disease Pub. Co., New York.

consideration of one group whose adaptation to food would seem to have been accomplished through the adjustment of the vegetative nervous system.

To make clear the course of reasoning which has led to this conclusion, it is necessary to review very briefly a few points regarding the vegetative nervous system and its action.

The nervous system is made up of the sensori motor and the vegetative systems. The latter, with which only we are concerned, is subdivided into the sympathetic and autonomic. The sympathetic reacts to small amounts of epinephrin. The autonomic is all that portion not included in the sympathetic, includes the vagus, and reacts to very small amounts of acetylcholen.[2] Under normal conditions these systems (the sympathetic and the autonomic) are in equilibrium, producing a "tonic innervation" of smooth muscles. Stimulation of one or the other system by hormones or other factors may produce contraction and spasm or paralysis.

Vagal stimulation, for instance, may cause turbulent gastric peristalsis which readily changes into retrograde peristalsis and may manifest itself in vomiting. Hence the benefit on cardiospasm and pylorospasm of atropin. If the motor vagus endings in the intestine are stimulated by pilocarpin or physostygmin, peristalsis is increased, and emptying of the bowel occurs. Under certain conditions, however, a tonic spasm of the sphincters may result, a hypertonicity with a spastic condition of the intestinal tract.[1] The opposite effect, a relaxation and quieting of the intestines follows the use of atropin, which paralyzes the vagus. Owing to its close relation to the glands of internal secretion, the autonomic nervous system doubtless has a marked influence, not only on the pancreas and the thyroid gland, but also on the entire mechanism of metabolism.[1]

In the present imperfect state of our knowledge regarding the effects of food on the infant organism, it is not strange that a certain number of infants react in an abnormal way to the food introduced into the body. A normal healthy infant fed on the breast takes its food at intervals with apparent satisfaction, does not vomit, has normal stools, and sleeps most of the time between feedings, grows in size and increases in weight regularly. A normal, healthy infant fed artificially usually reacts in a similar manner.

A number of apparently normal, healthy infants fed on the breast, take their food with seeming satisfaction, grow and increase in weight, but are unhappy and restless, cry more or less constantly, sleep only at intervals for short periods of time, vomit and are constipated.

2. W. H. Gaskell: The Involuntary Nervous System, Monographs on Physiology, Edited by E. H. Starling.

A much larger number of artificially fed than breast fed children react in this abnormal way and present this picture of unhappy, restless, sleepless, crying infancy. The cause of this state is to be sought in the manner in which the individual child reacts to its food. Where the fault lies is not readily discernible. Available laboratory methods by which the offending substance or combination of substances can be detected and removed from the diet are lacking. The correction by the usual clinical methods of experimentation with various foods, and their combinations, is a very slow one and one not always followed by success. It is to this group of cases, whether breast or artificially fed, that the present paper desires to call attention. The cases are fairly common, and extremely intractable unless treated in the manner to be referred to.

In this group are to be found: (*a*) the colicky baby; (*b*) the sleepless baby; (*c*) the restless baby; (*d*) the irritable baby which does not prosper on any food, although there is neither gastric nor intestinal indigestion; (*e*) the baby suffering from pylorospasm accompanied by vomiting, usually projectile in type; (*f*) the constipated baby.

Without exception these infants are of the spasmophilic diathesis; and usually with a neurotic ancestry.

That usual therapeutic procedures and rearrangement of diet are inadequate to relieve the symptoms is amply demonstrated by the fact that these cases drift from physician to physician to be relieved finally by time; that is, when the age is attained when solid food is used, or by operation when pyloric obstruction is diagnosticated.

The typical history is about as follows: Infant born at full term, entirely normal, fed at the breast, cries more than it should during the early days of life and is supposed to be having a great deal of colic. Beginning at once, or after several weeks, vomiting ensues; this is usually explosive but need not be of this type. The vomiting is not related to the taking of food and may occur after each nursing period, or only once or twice in twenty-four hours. The volume may be more than has been taken in at one nursing. Its very irregularity is characteristic. Because of this vomiting and irritability, changes are made in the diet, first, by modifying the breast feeding, in time, in quantity and by dilution. Usually this is ineffective. Artificial feeding is then begun and the condition becomes worse instead of better. The infant may gain normally, make only meager gains, or fail to gain at all. The infant is constipated, sleepless, and cries much more than it should.

These cases pass from hand to hand, changes are made in formula and method, but usually without much result. The diagnosis, where propulsive vomiting exists, is pylorospasm. And this is the correct diagnosis. Wherever the propulsive vomiting does not exist, the diagnosis is not made.

SYMPTOMATOLOGY

The sypmtomatology depends on when these children are first seen. Their weight is normal in the beginning; after some months they are

below normal and frequently rhachitic. The infants without exception are hypertonic. They are able to raise their heads at birth, and grasp objects firmly. They can support themselves on their legs at the end of six or eight weeks if held up. The entire body may be raised by the head because of the rigidity. They are often referred to by the nurse and family as being very strong. An abdominal examination often reveals active gastric or intestinal peristalsis. One obtains the impression that they are unusually alert, they cry more or less constantly, rarely sleep except in short stretches, and are not happy babies. If this condition has not been corrected before five or six months, the emaciation may be marked.

Cases of this type present, in the order of their importance:

1. *Vomiting,* which, as before mentioned, is characteristically irregular, often with visible peristalsis.

2. *Crying,* which is more or less constant and violent.

3. *Insomnia;* this is usually a striking symptom, and with the crying creates a household state bordering on demoralization.

4. *Constipation,* resistant to ordinary laxative and dietetic treatment, and distinctly spastic in type, the stool being soft after it has once started.

5. *Cold Extremities,* with pallor, cyanosis and subnormal temperature. This is met with only occasionally.

6. *Underweight;* the degree depending on the age at which correct treatment is instituted—under 8 weeks, 1 to 1½ pounds; from 3 to 6 months, 3 to 4 pounds.

TREATMENT

Correct therapy shows results in this condition which are unusual in their regularity and in the rapidity with which they occur. Where indigestion exists, this of course must be improved by appropriate methods of feeding; as a rule, however, there is no indigestion and no change in diet is necessary.

By the administration of atropin in sufficient doses the entire picture changes in from forty-eight to seventy-two hours; in some cases even in twenty-four hours. The method of administering the atropin is in 1:1,000 solution, beginning with 1 drop in each bottle of food and increasing it in forty-eight hours, if there is no improvement, to 2 drops in each bottle, and later to three or four or more drops, if necessary, with an interval of forty-eight hours between change in dosage.

If the diagnosis is correct, it will be found that the atropin controls the frequency of the vomiting. The child begins to sleep very much better and for longer periods; in its waking moments it is comfortable, and cries less or not at all. The vomiting usually ceases entirely or only occurs at rare intervals. Occasionally, one symptom of those enumerated persists long after the infant is in all other respects normal, and that is usually found to be due to some faulty detail in manage-

ment. As a rule 1 or 2 drops of atropin at each feeding are enough, although frequently 3 or 4 drops or more are necessary to obtain a result. At intervals the atropin is reduced in amount, then discontinued. If there is no recurrence of symptoms, it is no longer required; if, however, the symptoms return, then the atropin must be resumed until repeated testing has shown it to be no longer necessary. This may require months; in some cases six months.

There have been no ill effects noticed from these large doses of atropin in infants, and it is a striking fact that infants a few months of age are able to take doses of atropin which would cause toxic symptome in the adult.

If atropin is used early and in sufficient dosage the number of cases of pylorospasm which finally reach the operating table as cases of hypertrophic stenosis will be reduced to an unimportant minimum.

It is not to be supposed that all infants who vomit, or cry, or do not sleep, or are constipated will respond to this treatment. On the contrary, if the cause is elsewhere than in the autonomic nervous system, there will be no result.

DIAGNOSIS

How is it possible to make a positive diagnosis? It is not possible to make a positive diagnosis by clinical methods, but one can make a probable diagnosis when the combination of vomiting, insomnia, crying, constipation and, sometimes, subnormal temperature exists in the presence of hypertonicity and in the absence of faulty feeding. Atropin, administered in full dose, will definitely establish the diagnosis.

The maximum dose of atropin used in twenty-four hours was 1/15 grain. The maximum amount administered in a single dose was 1/16 grain, where a mother at the Welfare Station at Lebanon Hospital misunderstood directions and gave a teaspoonful of a 1:1,000 solution instead of 1 drop, to a baby under 6 months. There were no ill effects.

The earliest symptom of the physiologic effects of atropin in infants is a flushing which gives them the appearance of having fever, which sometimes actually exists, but is usually not high, dilatation of the pupils and slight puffiness of the lids.

In two institutional infants not belonging to this group, atropin in doses of 1 or 2 drops of 1:1,000 solution, after several doses produced hyperpyrexia, abdominal distention, flushing, restlessness, vomiting and diarrhea. This syndrome could be reproduced at will. Atropin in small doses paralyzes the efferent nerve endings which terminate in peripheral ganglia (for example, vagus) and in involuntary muscle. Atropin in large doses paralyzes the secretory fibers of probably all glands, including mucous glands, pancreas and liver; stops the movements and paralyzes the involuntary muscular fibers of the intestines so that they only contract feebly or not at all when directly irritated.[3]

3. Brunton: Pharmacology, Therapeutics and Materia Medica. Ed. 3, Lea Bros., Phila.

Atropin by virtue of the greater than normal transmission of blood through the tissues increases metabolism and results in an increased elimination of waste products.[4]

Perhaps the description by a nurse in charge of one of these infants (Case 1), written at my request, will give a better picture than could be obtained in any other way.

REPORT OF CASES

CASE 1.—Referred by Dr. George L. Brodhead. Edward R., born March 8, 1917; normal delivery; mother of nervous, apprehensive type; baby weighed 7 pounds 8 ounces. Breast feeding was started on the fourth day. The baby

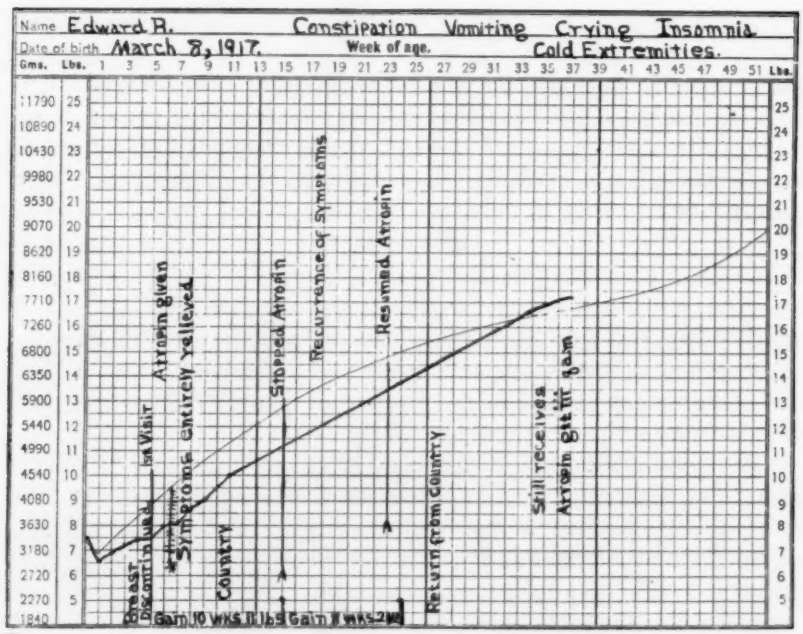

Chart 1 (Case 1).—Weight chart of Edward R.

obtained 1½ ounces on the fifth day every three hours. Meconium stools for three days. Castor oil, minims 10, produced a large mucous stool. The baby vomited mucus frequently for the first two and a half days. Lost 1.6 pounds by end of the first week. Temperature always between 98 and 98.2 F.

Second Week: The infant cried one to two hours in twenty-four; obtained on an average 2 ounces at each nursing; stools normal; gained 6 ounces. Weight 6 pounds 14 ounces.

Third Week: Cried two to two and a half hours in twenty-four; received 2½ ounces at each nursing; stool normal; three to four daily passed with a large amount of flatus and considerable distress. Gain, 5 ounces. Weight 7 pounds 3 ounces. Mother up part of the day now; very nervous and depressed (condition not caused by baby).

4. Bartholow: Materia Medica and Therapeutics. Ed. 11, Appleton and Co., New York.

Fourth Week: Baby cried as though in pain three to four hours daily. (*a*) Given barley water, ½ ounce before each three-hour nursing; (*b*) given two feedings of 5-20 whole milk, with 1 ounce lactose in twenty-four hours. Regurgitated about ½ ounce after three feedings daily. Vomited 1 to 4 ounces of sour curds after two feedings. Vomiting and regurgitation always occurred at time of stools and crying periods, which varied from day to day, most frequently occurring during the early morning or late afternoon. Weight increased 3 ounces. Weight 7 pounds 6 ounces.

Fifth Week: Breast nursing discontinued. (*c*) Baby given 3 ounces 6-20 whole milk; regurgitated 1 ounce of each feeding; cries six hours daily, frequently holding breath; colic (?); no relief obtained by gastric lavage, which showed a clean, sweet stomach two hours after feeding, or colonic irrigations, which were almost impossible to give as the anus became tense and contracted, then relaxing; no stool for twenty-four hours after starting bottle feedings.

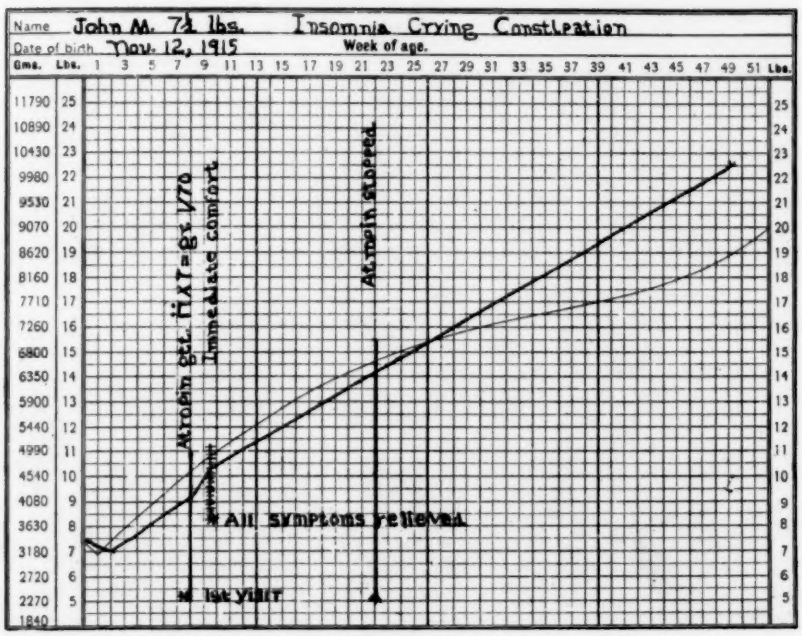

Chart 2 (Case 2).—Weight chart of John M.

Milk of magnesia, ½ dram daily, produced one to two large, digested stools. Feedings reduced to 2½ and then to 2 ounces; lime water in each feeding, with no change in amount or frequency of vomiting or regurgitation. Gained 1 ounce. Weight 7 pounds 7 ounces.

Sixth Week: (First visit.) (*d*) Milk increased to 14 in 28 ounces; 3 ounces every two and a half hours; regurgitation and vomiting not increased by more frequent or larger feedings; crying periods the same; finger tips and lips sometimes blue after hard cry; hands clinched constantly; body tense.

(*e*) Dextro-maltose substituted for lactose; no improvement.

(*f*) Same formula continued with 1 minim atropin sulphate 1:1,000 in each two and a half hour feeding. After the third feeding a marked change was noticed in the baby's condition; first feeding retained; second and third retained for two hours; the baby then vomited about ½ ounce of curds. It then passed a large normal stool; no laxative given. All feedings were retained on the

following day; the baby practically stopped crying; entire body comfortably relaxed; gained 3 ounces in weight.

Thirty-two hours after the first dose the baby's body felt hot; temperature 100.4 F.; face flushed, pulse 124. Atropin was omitted in the next feeding. Some puffiness under the eyes was noticed in the morning; temperature normal. Atropin 1:2,000, one-half drop, given during the day in each feeding, with some slight return of spasmodic crying and tension of body; atropin, 1:1,000 given in two succeeding feedings; condition relieved; gained 3 ounces. Quality and quantity of food unchanged. Atropin, 1:2,000 given in each feeding until the baby showed symptoms of returning tension and discomfort. Then 1:1,000 was given for several feedings, controlling the condition entirely. In five days the baby gained 9 ounces; temperature 98.6 to 99 F. There was one large, digested stool daily, though still being passed with great effort and

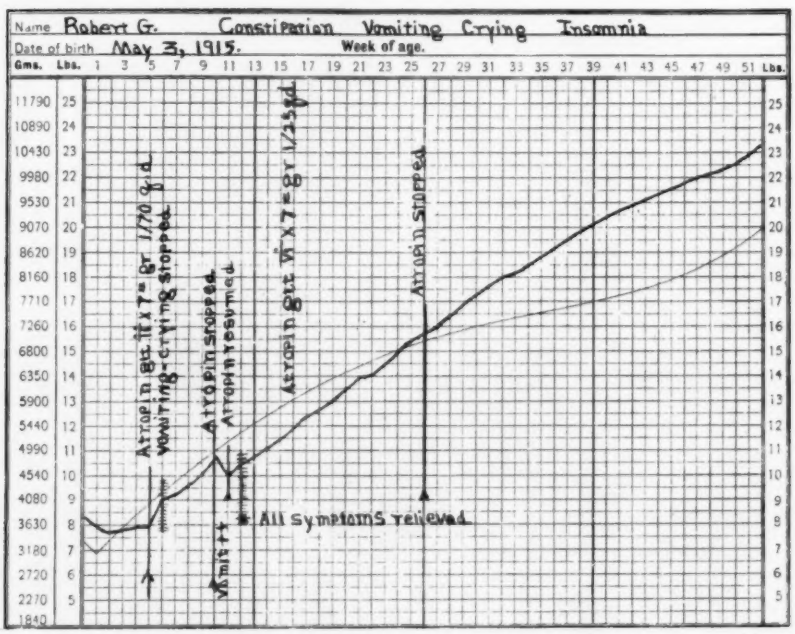
Name Robert G. Constipation Vomiting Crying Insomnia
Date of birth May 3, 1915. Week of age.
Gms. Lbs.
Atropin gtt. ii x 7 = gr 1/70 q.i.d.
Vomiting-crying stopped.
Atropin stopped.
Atropin resumed
Atropin gtt. iii x 7 = gr 1/215 q.i.d.
Atropin stopped.
All symptoms relieved.

Chart 3 (Case 3).—Weight chart of Robert G.

some crying. The baby's whole expression changed; it looked comfortable; slept most of the time; extremities warm. From birth until starting the atropin treatment the baby's hands and feet were always cold unless especially covered with extra blankets and with hot water bottles in the crib.

Chart 2 (Case 2).—John M., born Nov. 12, 1915; weight 7½ pounds. Complaint, insomnia, crying and constipation. Jan. 12, 1916, first visit; aged 2 months; weight 9½ pounds; first baby; labor normal.

Diet: (1) Breast milk; (2) milk, limewater, lactose, water; (3) condensed milk; (4) malt soup.

New Diet: Imperial granum, water, maltine, whole milk; 2 drops of atropin in each bottle. Ten days after new régimen was started there was a gain of 1 pound; general condition + +.

Oct. 26, 1916, aged 11 months; weight 22½ pounds; stopped atropin at 5 months; stopped maltine at 9 months. The child did well after atropin was begun.

Chart 3 (Case 3).—Robert G., born May 3, 1915; physical condition apparently good; weight 8½ pounds. Complaint, constipation, vomiting, crying and insomnia.

May 15, 1915, first visit, aged 12 days; weight 7 pounds 13 ounces.

Feeding: (1) Breast milk; (2) breast feeding and sodium citrate; (3) malt soup; (4) Schloss milk; (5) modified cow's milk. Paregoric was used for the crying and sleeplessness, without result.

June 15, 1915, atropin, 2 drops in each feeding; weight, 8.1 pounds.

June 22, 1915, weight 8.15 pounds; symptoms absent.

July 15, 1915, atropin stopped four days previously with return of symptoms.

Aug. 27, 1915, 4 months old; weight 11.14 pounds; occasional vomiting.

Dec. 4, 1915, 7 months old; weight 17.4 pounds; atropin stopped at sixth month; no symptoms.

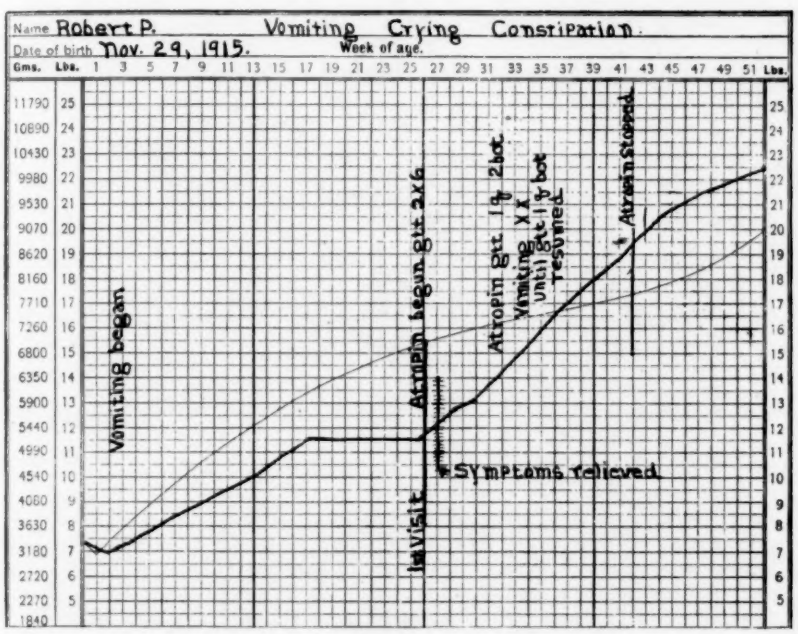

Chart 4 (Case 4).—Weight chart of Robert P.

Chart 4 (Case 4).—Robert P., born Nov. 1, 1915; weight 7.5 pounds; second child; labor normal. Complaint, vomiting, crying and constipation.

Feeding: (1) Breast and condensed milk two and a half months; slight gain; no vomiting; (2) top milk, granum, malt soup; curds, mucus and diarrhea; (3) barley, with skimmed milk; irregular gain; (4) granum, evaporated milk, lactose; (5) aqua cakes, water, lactose, milk. Began to vomit at three weeks (projectile), and has continued up to the present.

May 25, 1916, first visit; aged 6 months; weight 11.10 pounds; atropin, 2 drops in each bottle. New Diet: Granum ½ ounce, maltine ½ ounce, milk 20 ounces, water 16 ounces, divided into six bottles.

May 31, 1916, weight 12.3 pounds; symptoms absent.

July 24, 1916, occasional projectile vomiting; weight 16 pounds; 8 months old.

Several attempts to stop atropin resulted in a recurrence of symptoms within forty-eight hours. At the age of 25 months the weight was 29½ pounds; height 34 inches.

Chart 5 (Case 5).—(Referred by Dr. A. W. Roff.) George H. S., born March 24, 1915; birth weight 9 pounds.

Complaint, insomnia, constipation, vomiting, crying and visible peristalsis. Second child; labor normal; present weight 8.15 pounds.

Feeding: (1) Breast three weeks; (2) condensed milk; (3) Horlick's malted milk; (4) granum; (5) modified milk; first visit (6) modified milk, fat 2½, protein ½, granulated sugar 6; May 12, 1915, (7) modified milk, fat 1¾, protein 1¾, dextro-maltose 6; 3 ounces every two and a half hours.

May 18, 1915, stool improved; vomiting severe; other symptoms not improved; weight 9.8 pounds; atropin started, 5 drops before each feeding.

May 19, 1915, no vomiting; quiet, but constipated; lips and throat dry; atropin reduced to 2 drops.

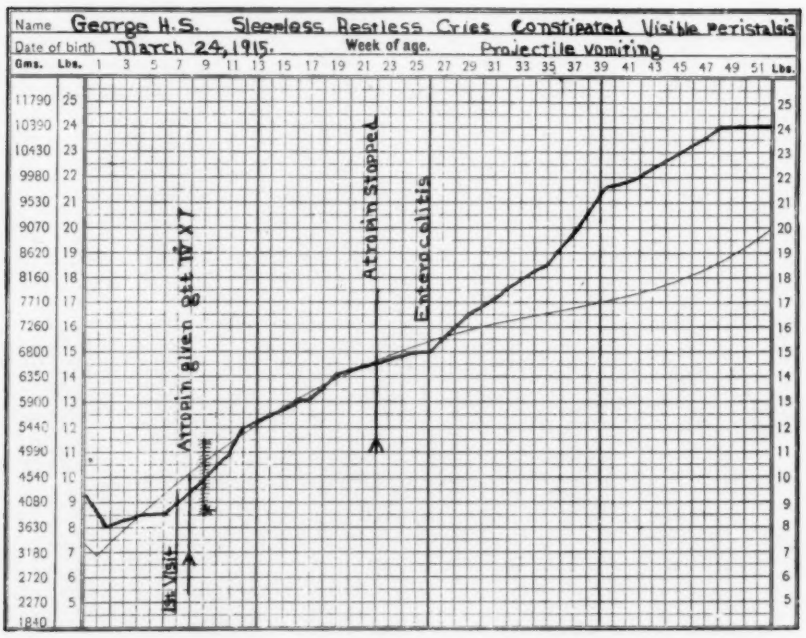
Name George H.S. Sleepless Restless Cries Constipated Visible Peristalsis
Date of birth March 24, 1915. Week of age. Projectile vomiting
Gms. Lbs.
Atropin given gtt IV X7
Atropin Stopped
Enterocolitis
Dr. Visit

Chart 5 (Case 5).—Weight chart of George H. S.

June 26, 1915, appearance, good; slight sign of rickets; vomits infrequently; sleep improved but cries and is constipated. Atropin stopped and paregoric, 12 drops, given in each feeding. This apparently had no effect. All symptoms returned. When atropin was used, it seemed to warm and quiet him.

July 23, 1915, weight 13½ pounds.

Formula: Milk 26, barley water 8, limewater 2, dextro-maltose 2½; 6½ ounces, seven feedings. Received atropin, 4 drops at each feeding; could not get along on less; peristalsis visible at times; other symptoms practically absent, excepting those of rickets, which were slow in clearing up.

Atropin taken more or less constantly until the child was 1 year old.

Chart 6 (Case 6).—(Through courtesy of Dr. W. M. Stone.) Grace G., born May 19, 1916; second child; first died in labor; birth weight 5.8 pounds. Complaint, irritability, crying, vomiting and slight constipation.

Feeding: (1) Breast, two weeks; weight dropped to 4.10 pounds the first week; (2) barley water, limewater; present formula (3), milk 7, limewater 1,

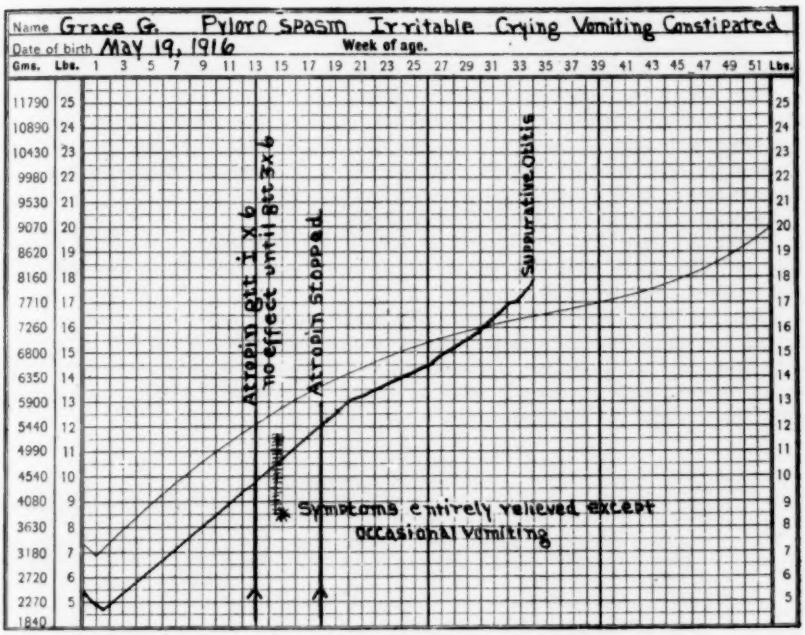
Name Grace G. Pyloro Spasm Irritable Crying Vomiting Constipated
Date of birth May 19, 1916
Week of age.
Gms. Lbs.
Atropin gtt i x 6 no effect until Bit 3x6
Atropin stopped
Suppurative Otitis
Symptoms entirely relieved except occasional Vomiting

Chart 6 (Case 6).—Weight chart of Grace G.

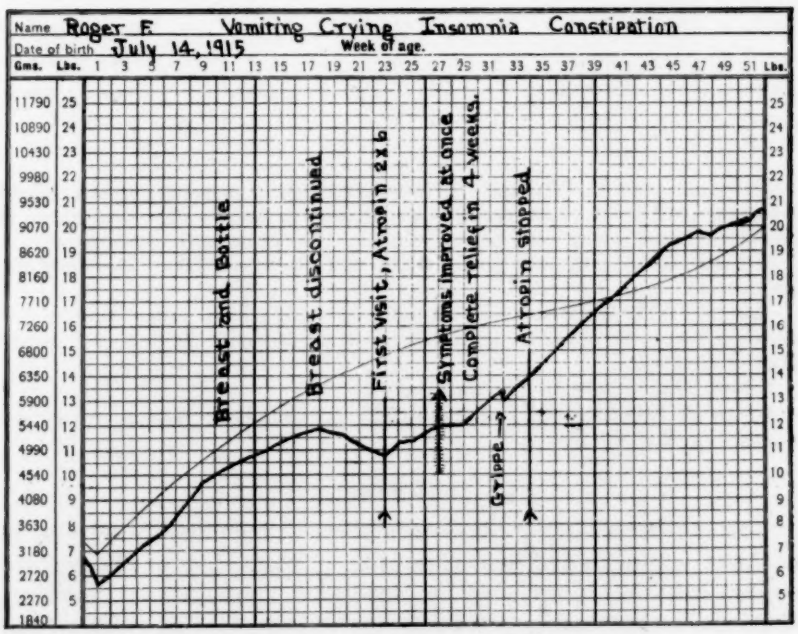

Chart 7 (Case 7).—Weight chart of Roger F.

cane sugar 2, barley water 12; 4 to 6 ounces every four hours; peptonized; peanut oil 1 drop fifteen minutes before feeding.

First visit, Aug. 16, 1916, weight 9.6 pounds; pale; abdomen shows peristalsis; stands with support; atropin, 1 drop in each feeding.

Aug. 25, 1916, symptoms relieved entirely, excepting the vomiting, which was not entirely eliminated; no effect from less than 3 drops of atropin solution.

Formula: Milk 12, dextro-maltose 2, limewater 1, barley water 10.

Sept. 1, 1916, milk 15 ounces, dextro-maltose 2, limewater 1, barley water 9. Vomits about once a week; other symptoms absent.

Infant at present is peculiar and unsocial.

Chart 7 (Case 7).—(Referred by Dr. J. T. Simonson.) Roger F., born July 14, 1915; birth weight 6.10 pounds. Complaint, vomiting, crying, insomnia and constipation; no gain recently.

Previous Feeding: (1) Breast; (2) breast, barley water (3 months); (3) breast, top milk 9½, dextrinized barley water 10½ (fourth month); (4) top milk 8 ounces; barley water; (5) top milk 24 ounces; (6) skim milk; (7) milk, sugar, water; (8) condensed milk 1:10; (9) top milk mixture peptonized; (10) malt soup; (11) protein milk.

SUMMARY OF CASES

Case	Name	Constipation	Vomiting	Insomnia	Crying	Peristalsis	Losing	No Gain	Under Wt., Pounds	Age When First Seen	Atropin Begun	Atropin Stopped
1	George S.	+	4th wk.	+?	+	+	..	+	1	7 wks.	5/ 8/15	8/24/15
2	Robert G.	+	2d wk.	+	+	..	+	..	1	5 wks.	6/ 8/15	11/ 5/15
3	Roger F.	+?	Early	+	+	..	++	..	4	5½ mos.	12/23/15	3/18/16
4	John M.	+	.....	+	+	..	..	Slow	1	8 wks.	1/12/16	4/16/16
5	Robert P.	+	3d wk.	+	..	+	..	+	4	6 mos.	5/26/16	10/ 9/16
6	Grace G.	+	8th wk.	+?	+	..	..	Slow	3	13 wks.	8/16/16	10/22/16
7	Edward R. ...	+	4th wk.	+	+	+	..	+	1½	5 wks.	5/24/17	11/27/17

First visit, Dec. 23, 1915, aged 5½ months; weight 11 pounds; marked rickets. Prescribed full milk, 20 ounces, granum ½ ounce, maltine ½ ounce, water 16 ounces, 9 ounces every four hours; atropin, 2 drops in each feeding.

Dec. 28, 1915, appearance good; sleeps well; is comfortable; some spitting; constipation.

Jan. 28, 1916, symptoms entirely relieved; only slight gain. Weight 9.12 pounds. Was now put on a straight milk formula: milk 30, lactose 1, barley water 5, limewater 2.

Feb. 5, 1916, weight 12.10 pounds.

March 28, 1916, aged 8½ months; weight 15.8 pounds; no symptoms; atropin stopped ten days previously.

SUMMARY

The hypertonic infant shows a tendency to rigidity, overaction of involuntary muscles and soon after birth develops signs of physical and psychic irritation, presenting the syndrome (physical) of vomiting, often visible peristalsis, constipation and imperfect nutrition; (psychic) of general irritability, insomnia and crying.

The etiologic factor of this state would appear to be a hyperexcitability of the autonomic nervous system.

Although apparently a feeding problem, changes in method and character of feeding does not correct the condition.

Atropin in doses of 1 drop or more of a 1 : 1,000 solution with each feeding usually relieves the condition at once and apparently increases the infant's tolerance for milk.

Atropin in these cases may be used in large doses without danger.

Investigation along these lines may reveal the etiology of hypertrophic pyloric stenosis.

666 West End Avenue.

A STUDY OF THE EFFECT OF HUNGER ON NERVOUS IRRITABILITY *

JESSE R. GERSTLEY, M.D., AND R. G. HOSKINS, M.D.
CHICAGO

During a summer vacation one of us (R. G. H.) observed that a fat, lazy, overfed dog, after having been lost for three days, returned thinner, brighter and more alert and active in every way. At this time A. J. Carlson in his work on the physiology of hunger suggested that gastric contractions were associated with some increase in general nervous irritability. Zybell's[1] clinical studies on spasmophilia also have a bearing on this subject. He concluded that any prolonged hunger aggravates the condition. He cast doubt on the idea that spasmophilia might be attributed to any specific element in the milk mixture, as the whey, the carbohydrate, or the starch additions, and demonstrated that independent of the nature of the food, a period of hunger prolonged over a few hours made the child decidedly worse. Indeed, he went even farther by showing that in spasmophilia the electrical reactions vary sometimes 1 to 2 milliamperes from morning to evening. These differences are due to the effect of hunger. The child, going through the night with very little food, awakens in the morning with pathologic reactions. After the day's feeding electrical irritability becomes normal. By changing the routine, by feeding through the night and giving very little food during the day, Zybell was able to reverse the reaction.

From these studies and observations one is tempted to advance the hypothesis that there is a general law in nature making increased nervous irritability coincident with increased hunger. If this were so, it could possibly be demonstrated in mathematical form by means of the electric current. This we attempted. Our first work was with infants, but the tendency of the electrodes to burn the skin, the restlessness of the children making accurate readings difficult, and our own reluctance to subject the children to any prolonged starvation, led us to transfer the work to dogs.

Perhaps the most extensive researches in animals on the subject of electrical excitability are those of Noel Paton[2] and his associates. These observers, in the course of their work on parathyroid tetany, demonstrated (*a*) the extreme variability of the reactions, and (*b*) the sensitizing effect of the current, namely, that a nerve after once

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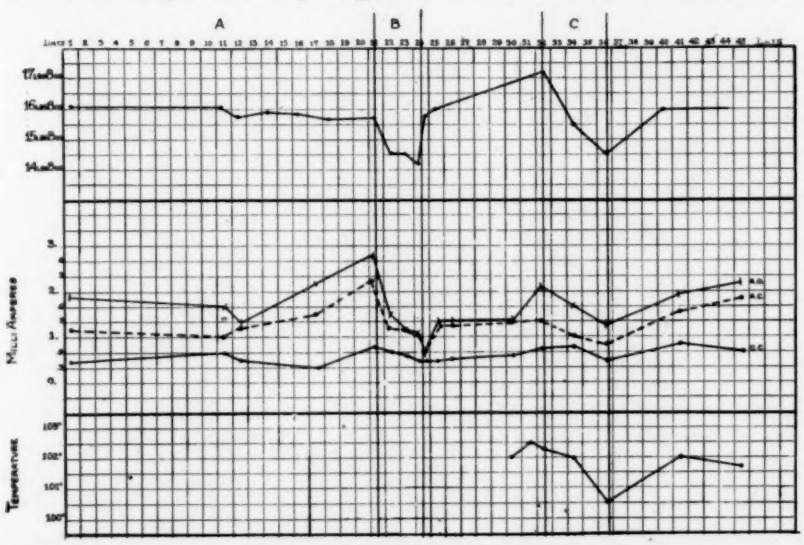
* From the Departments of Pediatrics and Physiology of the Northwestern University Medical School, and the Sarah Morris Hospital for Children.

1. Zybell: Jahrb. f. Kinderh., 1913, **28,** 29.
2. Paton, N.: Quart. Jour. Exper. Physiol., 1916, **10,** 203.

reacting becomes sensitized, and then reacts to about one-half to one-quarter of the primary stimulus.

The problem which we thought could easily be settled in a few weeks lengthened to almost two years. During this time we learned much about electrical reactions, particularly the difficulty of obtaining *any sort* of definite standard. In a normal dog, even at best, the reactions show extreme variability, changing not only day by day, but sometimes almost minute by minute. During moist days the atmosphere seemed to induce currents in the electrical machine; on other days small deflections in the needle could be traced to the iron work in the building, electrically charged for some unknown reason. To hold animals quiet for any length of time is difficult, and one is often



Period A, change of hygiene of kennels; Period B, three days' starvation; Period C, four days' starvation. AO, anode opening; AC, anode closing; CC, cathode closing. The readings were all taken in the morning, except on the twenty-fourth day when a. m. and p. m. observations were made.

confused as to whether the muscular contraction is due to the current, to shivering, or is voluntary. Then again, dogs vary in temperament, some being exceedingly nervous, irritable and timid, while others are calm and make quiet subjects. Due to these many and often uncontrollable variables, we threw out repeatedly whole series of observations; at times we even despaired of obtaining any definite result. A few experiments were carried out successfully, however, and the accompanying curve may be taken as illustrative of some of the series.

The technic of these experiments was that ordinarily employed in obtaining the reactions in children. In order to be sure that the animals were experiencing the true hunger effects, we controlled their weight and temperature.

DISCUSSION

In studying the chart, particularly AOC, which observers have found the most constant, one finds some increase in the electrical excitability during the hunger periods, but we have learned of so many variables, and have ploted so many curves which do not show this increase, that we do not feel justified in asserting this as a definite law. That there is no direct *parallelism* between electrical excitability and duration of the hunger period, or between the former and the weight curve, however, is certainly shown by the p.m. reading of the twenty-fourth day. Here the animals had been on a full diet for six hours; the weight curve took a sharp rise, but the curve of electrical excitability went down. At no time, in spite of the severity of the hunger and the drop of weight, did starvation bring COC anywhere near 5 milliamperes. Thus we believe hunger to be of no great practical importance in predisposing a normal individual to spasmophilia. We believe the general subject of hygiene to be more important. During the first few days the dogs were confined in dark, unpleasant kennels; their appetites were poor and they were unhappy. At A they were placed in pleasanter quarters, were allowed freedom, fresh air, exercise and sunshine. It will be seen that during the period C, after four days' starvation, the reactions are barely lower than in the pre-starvation, poor hygiene days.

CONCLUSIONS

We were not able by means of the electric current to demonstrate in all cases a constant parallelism between the length of the hunger and the degree of *electrical* irritability. This latter seems to be influenced more by general hygiene, conditions of the kennels, exercise, sunshine, appetite and body weight than by the hunger factor alone. Perhaps the parallelism may be demonstrated by psycho-analytical methods. We certainly can confirm the work of Noel Paton and his associates as to the extreme variability of these reactions and the sensitizing effect of the electric current. This latter observation may be of clinical value. One should never be satisfied with a first reading. The observation should be repeated several times; often a contraction will be obtained by one half to one fourth the original stimulus. The failure of the COC even after four days' hunger nearly to approach 5 milliamperes leads us to conclude that hunger can scarcely be a factor in predisposing normal individuals to spasmophilia.

We wish to express our thanks to Dr. O. T. Schultz for the privileges of the laboratories of the Nelson Morris Memorial Institute, and to Mr. E. L. Berry of Northwestern University Medical School for his technical assistance.

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CLINICAL DEPARTMENT

THE TREATMENT OF ENURESIS WITHOUT DRUGS

WITH A REPORT OF THIRTY-FOUR CONSECUTIVE RECOVERIES *

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Enuresis may be caused by organic diseases and by malformations. The present discussion, however, has to do only with the incontinence of urine ordinarily seen in childhood.

In order to understand the treatment of this condition, it is necessary to know so far as possible the physiology of normal urination. This was described by Goltz[1] in 1874, and the general principles set forth by him at that time have continued to be accepted with little modification. Stated in its simplest terms, the act of micturition may be described as follows:[2] Gradual distention of the bladder induces rhythmic contraction of its walls. This contraction increases until a few drops of urine are expressed into the posterior urethra, causing a sensory stimulus, the afferent part of the reflex, which, passing to its nerve center in the lumbosacral cord, arouses the afferent impulses by which the bladder is made to contract vigorously and the sphincter to relax. This mechanism is, however, almost completely under the control of the will, so that micturition may be made to begin before the bladder is full, and, on the other hand, the action of the reflex may be prevented by a voluntary exercise of will power. This urinary reflex is of such a primitive character that it persists even after section of the spinal cord.

In infants, it can be excited by almost any irritation of the pelvic organs, thereby causing involuntary micturition.

Infants may begin to acquire control of this mechanism during their waking hours at the end of the first year. If they do not gain control during the third year, incontinence may be said to exist.

Enuresis in children is, then, not a disease, but simply the persistence of an infantile condition, or habit. Why this habit persists in

* Submitted for publication Jan. 15, 1918.
* Read at a meeting of the New England Pediatric Society, Boston, Dec. 14, 1917.

1. Goltz: Arch. f. d. ges. Physiol., 1874, **8**, 478.
2. Starling, E. H.: The Principles of Human Physiology, 1915.

one child and not in another has furnished a fertile field for speculation without furnishing a satisfactory solution.

The following affections have been considered possible causes of this condition:

Anemia	Tuberculosis	Distant organs as the nose, throat and eye
Malnutrition	Enteritis	
Heredity	Rheumatism	Adherent prepuce or clitoris
Neurasthenia	Malaria	Hyperacidity of the urine
Chorea	Syphilis	Alkalinity of the urine
Epilepsy	Uric acid diathesis	High specific gravity
Hysteria	Rachitis	Low specific gravity

The mere enumeration of this large number of affections is evidence that the real cause has not been found. In this series of cases the treatment of no one of these affections mentioned has relieved enuresis.

In a group study of 100 children reported by the writer[3] during the present year, no special incidence was found of enuresis with adherent prepuce or clitoris, mental retardation, carious teeth or malnutrition. There was, however, marked incidence with nasopharyngeal obstruction. These observations are verified by the study of a second group of 100 children which give almost identical results.

Many drugs have been used for the treatment of enuresis, but no specific has been discovered. Those most frequently given are:

Citrate and acetate of potash	Hexamethylenamin	Bromids
	Strychnin	Thyroid extract
Benzoate of soda	Hyoscyamus	Belladonna

The children included in the series of thirty-four cases presented in this paper all recovered without the use of any drugs whatsoever.

In discussing the following method of treatment the expressions "wet habit" and "dry habit" are used. By "wet habit" is meant the infantile condition where the reflex acts more or less normally, but the mind of the child is unconscious of the sensory impulse or else disregards it. By "dry habit" is meant the child perceives the sensory impulse and his will acts to control the reflex. The following treatment is directed toward teaching the child the "dry habit" and, on the other hand, of interrupting and eliminating the "wet habit."

This treatment may be considered to consist of four methods, all or one of which may be used in a given case to establish the "dry habit." The "dry habit" once inaugurated must then be continued by careful oversight.

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3. Emerson, William R. P.: A Group Study of the Results of One Hundred Physical and Mental Examinations of So-Called Well Children, Internat. Clin., **11,** Series 27.

First Method.—Mental suggestion. This consists of arousing the patient's interest and attention to the act by rewards and other mental suggestions. This method of treatment is entirely adequate to relieve about 40 per cent.[4] of all cases of enuresis. It is this mild class of cases so relieved that are supposed to have reacted to the use of drugs, particularly to atropin, because in giving atropin, the patient is seen repeatedly, and, more or less unconsciously suggestion is used over a considerable period of time.

TABLE 1.—SHOWING INCIDENCE OF EIGHTEEN CASES OF ENURESIS WITH OTHER CONDITIONS, OCCURRING IN A GROUP OF 200 CHILDREN

No.	Patient	Naso-pharyngeal Obstruction	Mouth Breathers	Underweight	Adherent Prepuce	Mental Retardation	Carious Teeth
6	M. H.	+	+	..	..	+	+
13	S. M.	+	+	..	+	..	+
7	F. R.	+	+	..	..	..	..
12	B. R.	+	+	+	..	+	+
8	R. C.	..	+	..	..	..	+
34	J. M.	+	+	..	..	+	..
11	G. K.	..	..	..	.	..	..
9	M. H.	+	..	..	.	..	..
10	E. A.	+	..	..	..	..	+
15	H. McK.	+	..	+	+	..	..
16	F. S.	+	+	.	..	..	..
19	I. S.	+	+	..	..	..	+
18	L. Y.	+	+	..	+	..	..
19	T. S.	+	+	..	..	+	+
20	N. S.	+	+	+	..	..	+
21	O. S.	+	+	..	..	..	+
22	E. S.	+	+	..	..	+	+
23	F. B.	+	+	+	+	..	+
		16 cases 88 per cent. (100 group 67 per cent.)	14 cases 77 per cent. (100 group 62 per cent.)	4 cases 22 per cent. (100 group 22 per cent.)	4 cases 22 per cent. (100 group 19 per cent.)	5 cases 28 per cent. (100 group 40 per cent.)	11 cases 61 per cent. (100 group 64 per cent.)

Second Method.—Establishing the Dry Habit. This is accomplished by teaching the reflex to act at regular intervals, and always before the time the "wet habit" occurs. In this way the patient is always dry. The periods of urinations are gradually lengthened. By such systematic training the mechanism of urination soon acts under

4. True in a series of eighteen cases of enuresis occurring in a group of 200 children.

the patient's control and the "dry habit" is established. This method is described by Newlin[5] as "The simple procedure of anticipating the involuntary by a voluntary emptying of the bladder."

Third Method.—Local irritation. This is brought about by irritating the vesicular sphincter and the posterior urethra by means of a bougie-a-boule, so that sensory impulses passing to the brain will become intensified sufficiently to attract the patient's attention, until the habit of control is attained.

A bougie-a-boule should be used under antiseptic conditions. It should be introduced along the posterior urethra into the bladder without pain. It should be withdrawn through the sphincter, then passed in and out about four times, sufficiently to cause a sensation of discomfort during the next few urinations. This sensation of discomfort in urinating brings the act into the child's consciousness, aiding him in control. The idea of this treatment was suggested by the report of a patient who was relieved after the passing of a sound, and also by the fact that most children with enuresis are entirely unconscious of the act of urination. They waken at night and find themselves wet.

Fourth Method.—Cerebrospinal irritation. This is produced by the injection into the spinal canal of physiologic sodium chlorid solution, producing such stimulation as to arouse the whole mechanism into consciousness when habit holds it under control. This procedure should be carried out exactly as for lumbar puncture. A small trocar should be used with cocain analgesia. Ten c.c. of spinal fluid are withdrawn and 12 c.c. of freshly prepared physiologic sodium chlorid solution introduced into the spinal canal. The child should then be kept in bed at least six hours. There is apt to be some reaction, namely, headache and nausea, unless the saline solution is prepared from freshly distilled water and then filtered. This method of treatment is necessary only in extreme cases of enuresis, or those complicated by mental deficiency. It should be used only after other treatment fails; even then the question arises whether lumbar puncture is justified for such a comparatively simple condition as enuresis. It should be borne in mind, however, that enuresis after puberty is not a simple condition. It affects that which most of all makes life worth living, association with other human beings. When combined with incontinence of feces, as in three of the cases reported, the question is not debatable. Good foster-homes, summer camps and schools refused certain of these children because of enuresis. The brothers and sisters of one boy turned against him because he was constantly

5. Newlin, Arthur: The Management of Enuresis, Arch. Pediat., 1916, **32**, No. 10.

wet with urine. To the sensitive child this condition of incontinence may almost amount to a tragedy.

These methods singly or combined, have been sufficient to relieve the thirty-four children in the following series. This series includes all children to whom there was opportunity to give complete treatment. For convenience of reference the cases are reported according to their reaction to these four methods.

REPORT OF CASES

GROUP 1.—Recovery brought about by mental suggestion. This group includes Cases 6 to 11, 13, 14 and 34—nine cases. These children presented nothing in their history out of the ordinary in such conditions.

Marion M., aged 5, No. 6, was being treated for vulvovaginitis, but the enuresis preceded this affection.

Fred R., aged 9, No. 7, was punished by an irate matron, with temporary improvement, but complete recovery was brought about by encouragement and rewards. This may be explained by the fact that rewards stimulate the attention, while punishment introduces the element of fear, which is repression.

GROUP 2.—Recovery brought about by establishing the "dry habit." This group includes Cases 18 to 25, 29 and 30—ten cases—four boys and six girls. These all failed to respond to mental suggestion. They were wet almost constantly at night. Cases 18, 22 and 24 were wet also during the day. All had been afflicted with this condition since birth. Their ages varied from 5 to 11 years. The three diurnal cases were sent to the toilet every hour during the first day and all cases every hour the first night until midnight, then every two hours until morning. The second day these periods were lengthened to one and one-half hours and the third day to two hours. In the majority of these cases the "dry habit" was established by the end of the week, the child going to the closet or using the chamber of his own accord when necessary. Four of these children had a certain hour when wetting occurred, two about one-half hours after being put to bed, and two children at 2:30 in the morning. These children were taken up shortly before these times, and it was necessary that this be continued longer than for those who did not have established periods for wetting. In the treatment of these cases an alarm clock was useful for wakening, especially for older children.

GROUP 3.—Recovery brought about by local irritation.

Lillian H., aged 13, No. 28 (B. Inf.),[6] had nocturnal enuresis all her life. A soft catheter, French, No. 15, was introduced into the bladder with little lubrication. It was then moved to produce slight irritation and withdrawn. Report four weeks later, no enuresis since treatment.

Hita F., aged 3, No. 31 (M. G. H.),[6] wet herself continually at night. A bougie-a-boule, size 9, was passed along the posterior urethra and through the vesical sphincter into the bladder sufficiently to produce irritation. The child reported at the end of a week, "I wet my bed no more." She asks for the chamber two or three times at night and keeps herself entirely dry. The mother of this child had enuresis until puberty.

Gertrude N., aged 14, No. 32 (B. Inf.), had enuresis since birth. A catheter, No. 12, French, was passed Nov. 19, 1917. Report thirteen days later, no enuresis since treatment. This patient had chorea in 1916.

Ernest H., aged 13, No. 33 (B. Inf.), had enuresis since birth. Even if awakened twice at night he would still be wet. Nov. 17, 1917, a bougie-a-boule No. 12 was passed. Twenty-seven days later the report was, no enuresis.

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6. B. Inf. Berkeley Infirmary: L. W. H., New England Home for Little Wanderers; M. G. H., Massachusetts General Hospital.

TABLE 2.—GROUP 1. MENTAL SUGGESTION

No.	Name	Age	Diurnal	Nocturnal	Recovered	Follow-up Report	Remarks
7	Fred R.	9	..	+	+	6 mos.	Liquids restricted; regular habits
6	Marion M. ...	5	..	+	+	6 mos.	Regular habits; pride; rewards; vulvovaginitis
8	Russ C.	9	..	+	+	1 yr. +	Liquid restricted; regular habits
9	Mary H.	16	..	+	+	1 yr. +	Regular habits
11	Gordon K. ...	7	..	+	+	1 yr. +	Regular habits
10	Ethel A.	7	..	+	+	1 wk.	Rewards; regular habits
13	Samuel M. ...	6	..	+	+	1 yr.	Regular habits
14	Edward R. ...	10	..	+	+	1 yr.	Regular habits
34	James M.	9	..	+	+	3 wks.	Regular habits

TABLE 3.—GROUP 2. "DRY HABIT"

No.	Name	Age	Diurnal	Nocturnal	Recovered	Follow-up Report	Remarks
18	Louis Y.	5	+	+	+	1 wk.	
19	Thelma S.	11	..	+	+	3 mos.	Vulvovaginitis
20	Norma S.	9	..	+	+	3 mos.	Vulvovaginitis
21	Ola S.	7	..	+	+	3 mos.	
22	Edna S.	6	+	+	+	3 mos.	
23	Freeman B. ..	6	..	+	+		
24	Lester B.	7	+	+	+	1 yr.	Thyroid-Saline 6 c.c.
25	Lillian H.	7	..	+	+	3 wks.	
29	Charlotte B.	5	..	+	+	15 mos.	Alarm clock 2 a. m.
30	Barbara B. ..	4	..	+	+	15 mos.	Alarm clock 2 a. m.

TABLE 4.—GROUP 3. LOCAL IRRITATION

No.	Name	Age	Diurnal	Nocturnal	Treatment	Follow-up Report	Remarks
25	Lillian H.	11	..	+	+ Cath. No. 12	3 wks. +	One treatment
31	Rita F.	3	..	+	+ Bougie No. 9	3 wks.	Mother had enuresis until puberty; one treatment
32	Gertrude M.	14	..	+	+ Cath. No. 12	13 days	Chorea 1916; 2 treatments
33	Ernest A.	13	..	+	+ Bougie No. 12	10 days +	Three treatments

In each of these cases this treatment was followed up by regular habits of urination, limited liquids and dry supper at night. As in Groups 1 and 2, each child was awakened at about 10 p. m. for urination and encouraged by mental suggestion to form the "dry habit." The report of this group is a preliminary one only, as it has been tried in too few cases to ascertain its true value.

GROUP 4.—Recovery brought about by introducing physiologic sodium chlorid solution into the spinal canal.

TABLE 5.—GROUP 4. SPINAL INJECTION

No.	Name	Age	Enuresis		Incontinence of Feces	Number C.c.*	Anesthetic	Reaction	Recovered	Report	Remarks
			Diurnal	Nocturnal							
1	John F.	6	+	+	+	10— 10+	None	None	+	6 mos.	
2	Francis H.	12	+	+	+	9— 10+	Ether	++	+	1 yr.+	"Psychopathic"; "erratic"; police
3	John M.	13	+	+	..	18— 10+	Ether	None	+	1½ yrs.	Refused summer camp
4	Thomas F.	4	+	+	..	10+	Ether	++	+	2 yrs.+	Mentally deficient
5	Francis McL.	7	..	+	..	10— 10+	Ether	None	+	1 yr.+	Lazy
12	Bertram R.	12	..	+	..	10— 10+	Ether	None	+	1 yr.	
15	Hector McK.	5	+	+	..	10— 10+	Cocain	+	+	2 wks.+	Mumps; adherent prepuce
16	Frank S.	4	+	+	..	10— 10+	Cocain	+	+	2 mos.+	Mumps
17	Irene S.	4	+	+	..	10— 10+	Cocain	+	+	2 wks.+	
26	Barnard W.	19	+	+	..	10— 12+	Cocain	None	+	6 mos.	Septum operation refused; lodgings
27	Hilda A.	11	+	+	+	10— 12+ 12+	Cocain	None	+	6 wks.+	Mentally deficient; refused sch'l

* The minus sign marks the amount of spinal fluid withdrawn and the plus sign the amount of saline solution introduced.

John F., Italian, aged 6, earned pennies by dancing in the public market. This boy had both incontinence of urine and feces. He was admitted to the Boston Dispensary Hospital for Children, April 4, 1913. The first night he passed both urine and feces in bed. The next day lumbar puncture was performed without anesthetic, 10 c.c. of spinal fluid being replaced by 10 c.c. of physiologic sodium chlorid solution. He had no more incontinence while in the hospital, eleven days, and was reported to have none six months later.

Francis H., aged 7, No. 2 (B. Inf.), reported by the Psychopathic Hospital as not mentally deficient, but erratic. He was truant and had trouble with the police. He had incontinence of both urine and feces. His bowels would move when he was 10 feet from the toilet without his paying any attention to the act.

He was wet almost constantly. In 1913, 10 c.c. of spinal fluid were replaced by 10 c.c. of physiologic sodium chlorid solution. He gained control of his bowels at once, but continued to have enuresis. Two years later the spinal treatment was repeated under ether. This treatment was followed by marked reaction, headache, vomiting and temperature of 103 F. The reaction passed off in twenty-four hours. It seemed probable that it was occasioned by the use of solution not absolutely fresh. He reported in 1917, no further incontinence since last treatment.

John M., aged 12, No. 3 (B. Inf.), had enuresis since birth. Wet every night. He had been given various treatments with no effect. Ten c.c. spinal fluid were replaced by 10 c.c. of physiologic sodium chlorid solution under ether. No reaction. Report eighteen months later, no enuresis since treatment.

Thomas F., aged 4, No. 4 (B. Inf.), was mentally deficient. Wet day and night, almost every hour. Ten c.c. of spinal fluid were replaced by 10 c.c. physiologic sodium chlorid solution. Marked reaction; headache; vomited five times; temperature 101 F. This reaction passed off in six hours. He reported after two years, November, 1917, no enuresis since treatment.

Francis L., aged 7, No. 5 (B. Inf.), had enuresis since birth. Ten c.c. of spinal fluid were replaced by 10 c.c. of physiologic sodium chlorid solution. Report eighteen months later, no enuresis since treatment.

Bertram R., aged 12 (L. W. H.), wet every night. Ten c.c. of spinal fluid were replaced by 10 c.c. of physiologic sodium chlorid solution. No enuresis while in the home, about four months.

Hector M., aged 5, No. 15, had enuresis every night. December, 1916, he was given "dry habit" treatment with good results. He was dry a whole week; then he was sent to the City Hospital with mumps and returned with a relapse of the enuresis habit. March, 1917, 10 c.c. of spinal fluid were replaced by 10 c.c. of physiologic sodium chlorid solution. There was some reaction for six hours, with headache and nausea. Since treatment the child goes to the toilet of his own accord. Report some months later, no enuresis.

Frank S., aged 4, No. 16 (L. W. H.), had enuresis day and night. He was treated by the second method with good result. January, 1917, he was sent to the City Hospital with mumps; returned wet every night. March, 1917, 10 c.c. of spinal fluid were withdrawn and 12 c.c. physiologic sodium chlorid solution were introduced. There was a reaction for about six hours, with headache and nausea. Report two months later, no enuresis.

Irene S., aged 4 (L. W. H.), had enuresis day and night. She wet herself during the day while sitting up in bed. She was taken to the toilet regularly, with no effect. Ten c.c. of spinal fluid were withdrawn and 12 c.c. of physiologic sodium chlorid introduced into the spinal canal. Some reaction occurred with nausea and headache for about six hours. Reported sixteen days following, goes to toilet of her own accord and has no enuresis.

Bernard W., aged 19, No. 26, was referred from the Psychopathic Hospital. Wet since birth; for the previous two years only two or three times a week. On account of his incontinence the average boarding house would keep him about four weeks. He considered himself a wanderer on the face of the earth and therefore earnestly desired treatment. His enuresis occurred regularly at about 2:30 in the morning. He was told to set an alarm clock at 2 a. m. This wakened the boarders, but not the patient. In April, 1917, 10 c.c. of spinal fluid were withdrawn and 12 c.c. of physiologic sodium chlorid solution introduced. Report three months later, no enuresis.

Hilda A., aged 11, No. 27, was referred from Jamaica Plain Children's Aid Society. Mentally deficient; incontinence of both urine and feces since birth; wet herself every one-half to three-fourths of an hour; expels gas from bowels without control; discharged from school on this account. July, 1917, 10 c.c. of spinal fluid were withdrawn and 12 c.c. of physiologic sodium chlorid solution introduced. Reported a month later, no incontinence of either bladder or bowels since treatment.

Just how the saline solution acts on the reflex in these cases is not clear. The effect was apparently the same when given under ether, cocain or without anesthetic. In two cases of dry tap and in two cases in which only 6 c.c. of physiologic sodium chlorid solution were introduced into the spinal canal there was no apparent effect on the enuresis.

657 Boylston Street.

XANTHOCHROMIA OF THE SPINAL FLUID WITH COMPLETE COAGULATION IN TUBERCULOUS MENINGITIS *

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During the last fifteen years the occurrence of xanthochromia (yellow discoloration) of the cerebrospinal fluid has been noted by several observers. Sprunt and Walker[1] have recently collected 100 examples from the literature which showed this condition. They have divided the cases with this variety of spinal fluid into two main groups: (1) those in which the color is due to dissolved hemoglobin or its derivatives; these fluids, as a rule, do not coagulate spontaneously and contain only a small amount of globulin; they are usually associated with tumors of the brain in contact with the meninges, or tumors originating in the ventricles; (2) the larger and more important group which comprise those cases showing the so-called "Froin syndrome." The fluid in this group is transparently clear, yellow, coagulates spontaneously and contains a large amount of globulin; it may or may not show pleocytosis, and it gives no positive reaction for hemoglobin.

Reviewing the cases recorded up to 1912, Mix[2] finds that whenever a spinal fluid conforms to the second group described it indicates that there is an isolated sac in the spinal subarachnoid space which has been shut off from communication with the ventricles of the brain. This condition may be due to (1) a meningitis which had caused adhesions between the meninges and the surface of the cord, (2) to a pachymeningomyelitis of the conus terminalis, or (3) to a tumor which compresses intradurally or extradurally. It always signifies organic disease.

REPORT OF CASE

History.—C. B., a boy, aged 9 months, was admitted to the Babies' Hospital, Aug. 23, 1917. The parents and one other child were healthy. So far as could be ascertained, there was no tuberculosis in the family. The baby was born at term and weighed 9 pounds. Fourteen days before admission he had a little fever and was more restless than usual. Four days later the head was retracted and he was so drowsy that he had to be aroused for his feedings. There had been no vomiting or convulsions, but he had considerable twitching movements during sleep.

* Submitted for publication March 11, 1918.

1. Sprunt and Walker: Bull. Johns Hopkins Hosp., 1917, **28,** 80.
2. Mix: Surgical Clinics of Chicago, 1915, **4,** 317.

Examination.—On examination the patient presented the typical picture of a tuberculous meningitis. He lay in a condition of deep stupor and slept most of the time. Even when aroused he took no notice of his surroundings. The head was slightly retracted, the fontanel full, and there were automatic movements of the arms and legs on both sides. The eyelids were half open and the eyes rolled continually from side to side. There was a coarse lateral nystagmus and an inconstant strabismus. The pulse and respirations were very irregular. The abdomen was not retracted; there was a marked *'tâche cérébrale,* and the liver and spleen were palpable at the costal margin. The extremities were moderately spastic and all the reflexes were exaggerated. The von Pirquet skin test was strongly positive.

The leukocytes in the blood numbered 10,000 per c.mm. of which 39 per cent. were polymorphonuclears and 61 per cent. were lymphocytes. A spinal puncture was made and 15 c.c. of fluid removed under low pressure. The fluid was slightly turbid and of a light amber color, resembling urine. The cells numbered 250 per c.mm., 76 per cent. of these being lymphocytes. Within ten minutes after withdrawal the fluid coagulated to a jelly-like mass, to such an extent that the tube could be inverted without spilling the contents.

For the next three days the drowsiness and prostration gradually increased. The spastic condition of the arms and legs continued but became more marked on the left side. Finally, there was a marked weakness of the left side of the face and a constant external strabismus of the left eye. The temperature ranged between 99 and 100 F. There was increased coma until death on the third day.

Necropsy.—At the postmortem examination made by Dr. Martha Wollstein, the brain and meninges showed the typical lesions of a tuberculous meningitis. The dura and pia mater were everywhere congested. Numerous miliary tubercles were present along the surfaces of both cerebral and cerebellar hemispheres. The lateral ventricles were slightly dilated and their floors peppered with miliary tubercles. Over the base of the brain there was an exudate of serum and fibrin covering the lower surface of the cerebellum, pons, medulla and extending over the island of Reil on both sides, causing marked thickening of the basal pia. The thickening of the meninges was very marked over the cervical cord. There were adhesions between the pia and dura along the upper 12 cm. of the cord, which is all that could be examined. The pia-arachnoid membrane measured on section nearly 0.5 cm. in thickness.

REMARKS

From the clinical picture, the von Pirquet reaction and the cytology of the spinal fluid it was evident that a tuberculous meningitis was present. The yellow color and massive coagulation of the spinal fluid, however, indicated that an obstruction existed somewhere in the spinal subarachnoid space. The spine was carefully examined but no evidence of Potts' disease was found.[3] It was then inferred that a solitary tubercle of the cord or a tuberculous thickening of the spinal meninges[4] probably existed.

The tubercle bacilli were not found in the yellow spinal fluid during life, but at the necropsy a clear, limpid fluid was obtained from the ventricles in which the bacilli were easily demonstrated.

3. Bromer: Am. Jour. Med. Sc., 1916, **151,** 378.
4. Hanes: Am. Jour. Med. Sc., 1916, **152,** 66.

In this case the thickening of the cervical membranes and the adhesions were quite sufficient to cause the obliteration of the subarachnoid space at this level. The spinal fluid in the lower portion of the canal was thus shut off from communication with the fluid in the ventricles above, and it is in similar conditions that the spinal fluid becomes yellow in color and coagulates spontaneously on removal.

I am indebted to Dr. L. Emmett Holt for the privilege of reporting this case.

PYLOROSPASM AND CONGENITAL PYLORIC STENOSIS

PROBABLY THE EARLIEST REPORTED CASE *

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In 1788 Hezekiah Beardsley, an American physician, published a report of a "Case of Scirrhus in the Pylorus of an Infant," which Professor William Osler, in 1903, identified as "congenital hypertrophic stenosis of the pylorus." This case report was republished under its newer title in *Archives of Pediatrics* (1903, 20) with the following note by Professor Osler:

> Cantley and Dent in a recent paper (*Lancet*, Dec. 20, 1902) state that the first record of this disease, which is now exciting a good deal of interest, dates back to 1841. The report here given by Dr. Beardsley of a very clearly and accurately described case, is, I think, worth republishing. It appears in the earliest volume of medical transactions issued in this country entitled, "Cases and Observations of the Medical Society of New Haven County in the State of Connecticut," New Haven, J. Meigs, 1788.

While Beardsley's monograph still stands as the first separate publication dealing with pyloric spasm and stenosis, the distinction of describing and actually naming with the term we now use the symptoms and pathology of this disease of the new-born apparently belongs to George Armstrong, an English pediatrist, whose work appeared in 1777, a textbook entitled "An Account of the Diseases Most Incident to Children from their Birth till the Age of Puberty, with a Successful Method of Treating Them, etc." (London, 1777). This textbook was accorded several reprintings, and was rewritten by Buchan in 1808. Clinical and post mortem findings, a typical case history, and a family history, including three similar cases, led Armstrong to conclude that a child which he had treated for "watery gripes" died from *"spasm of the pylorus."*

On page 49 of the edition of 1777 Armstrong, in discussing the reasons why antimonial emetics sometimes fail to produce beneficial results in intestinal diseases, has described a macerated or softened condition of the intestines seen in some fatal cases. He says:

> In a child about 3 weeks old that died of the watery gripes, and which I opened some time since, I found most of the stomach toward the upper orifice and almost the whole fundus in the same tender state. But toward the pylorus the structure was firm enough as likewise that of the intestines, both small

* Submitted for publication March 22, 1918.

and great. The stomach was quite distended with curdled milk and victuals with which the nurse had crammed the child, mixed likewise with some of the chalk julep, but the whole intestines were remarkably empty. There were no morbid appearances to be observed anywhere but in the stomach, and this viscus being so full while the intestines were almost empty, it looked as if the disease had been chiefly owing to a *spasm in the pylorus,* which prevented the contents of the stomach from passing into the duodenum.

Perhaps cases of this kind are more frequent than is commonly imagined. . . . What is remarkable, this is the third child (and they had never had any more) which the parents have lost at the same age and with the same disease. And this was likewise the case in the family where the other died.

As to his term "the watery gripes," we find on page 20 that "vomiting and green stools" are the threatening symptoms which if allowed to continue "often end in convulsions and death."

The age of the child, "about three weeks," shows the development of vomiting during what we now recognize as the conventional period for the beginning of the disease. The citation of three other cases, two of them in the same family, occurring at the same period of life, shows that Armstrong believed that he had discovered a hitherto undescribed condition, though he says, very modestly: "perhaps cases of this kind are more frequent than is commonly supposed."

The stomach of a soft and flabby texture in contrast with the firm pylorus, and containing food "while the whole intestines were remarkably empty" leads him logically to his conclusion that "it looked as if the disease had been *chiefly due to a spasm in the pylorus.*" He, however, omitted any comment on the intimate structure of the pylorus itself, being probably content with a physiologic rather than an anatomic explanation.

In the case reported by Beardsley as "Scirrhus in the Pylorus of an Infant," death occurred at the age of 5 years, after a history of malnutrition and vomiting beginning with the first week of life. The stools in Beardsley's case had always been "small in quantity and of an ash color," that is, free from bile pigment. Moreover, the gallbladder was "nearly five inches in length and more than one in diameter, ... its contents were rather solid than fluid and resembled flesh in a highly putrid state." Cholecystitis is not a usual condition in pylorospasm or pyloric hypertrophy of infants, and aside from Armstrong's priority of publication by eleven years, Beardsley's case is in this respect at least less true to type than Armstrong's.

In may not be out of place to add that George Armstrong organized the first and only hospital and dispensary for poor children in London in his day in the year 1769. It was here he made his therapeutic experiments and had opportunity to compare clinical records with the post mortem findings scattered so plentifully through his textbook. Here, too, he formulated his excellent "Rules for Nursing." It is a

sad commentary on the spirit of his day that after spending practically all his time and savings in building up the dispensary, he was forced to close it in the year 1781 for lack of financial support, although in the twelve years of its existence over 35,000 children had received treatment. This keen observer, brilliant writer and pioneer physician-philanthropist, who was blessed with the true scientific spirit and an almost prophetic outlook, died in obscurity, unpraised and unrewarded excepting in the grateful remembrance of the poor of London.

1726 M. Street N.W.

CONGENITAL INTESTINAL OBSTRUCTION, WITH THE REPORT OF A CASE*

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Although the standard works on embryology, anatomy, pathology and pediatrics seldom refer to the interesting condition of congenital obstruction of the small intestine, isolated case reports are not infrequent. In the large necropsy service of Bellevue Hospital during the past fourteen years, congenital obstruction of the intestine was encountered twice only.

Louise Cordes[1] collected fifty-six cases from the literature up to 1901, and reported a case of her own. Ballantyne,[2] in discussing the condition, says: "Antenatal narrowing or occlusion of the intestine is not very rare, but being an internal malformation, doubtless often escapes discovery." He reported a case with which he was acquainted and mentions the cases published by Durante[3] (two cases), Jackson,[4] A. Katz,[5] and Kirmisson,[6] not included by Cordes. Kirmisson's patient had been operated on for anal atresia soon after birth "with apparent success;" however, the infant died and necropsy showed a valvular obstruction of the duodenum, in addition to the anal defect. Spriggs[7] gives an interesting account of twenty-four cases, with an excellent discussion and extensive references to the literature. Other cases of this condition as well as cases of congenital obstruction of the jejunum and cases of multiple obstructions have been reported. In an extensive search of the literature I have found a total of 143 cases of all kinds of congenital obstructions of the small intestine. The reader is referred to the appended list of references for details of these cases.

Kaufmann[8] asserts that atresia of the small intestine is especially common at the point where the common duct joins the duct of Wirsung at its entry into the duodenum. Combinations of duodenal atresia with

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* From the Pathological Department of Bellevue and Allied Hospitals, Dr. Douglas Symmers, Acting Director.

1. Cordes, L.: Arch. Pediat., 1901, **18,** 401.
2. Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene, 1905; Tr. Edinburgh Obst. Soc., 1892, **17,** 133.
3. Durante: Bull. Soc. anat. de Paris, 1901, **76,** 593.
4. Jackson, J. B. S.: Boston Med. and Surg. Jour., 1858, **59,** 355.
5. Katz, A.: Bull. Soc. anat. de Paris, 1901, **76,** 471.
6. Kirmisson, E.: Traité des maladies chirurgicales, 1898, p. 412.
7. Spriggs, N. I.: Guy's Hosp. Rep., 1912.
8. Kaufmann, Eduard: Spezielle pathologische Anatomie, **1,** 460, 6th ed.

defective development of the esophagus and with atresia of the rectum have been observed. Drennan and Clark[9] state that but one point of actual closure usually exists, but that, in addition, one or more points of stenosis may be found.

Productive fetal peritonitis and enteritis, thrombosis of the mesenteric vessels, anomalies in the development and distribution of the blood vessels, volvulus, intussusception and anomalies of the yolk stalk have been given as the possible causes. Rowland[10] reported one case and mentioned four others which, at operation, showed a complete twist of the mesentery of the small intestine. He also says that inspissated meconium may cause complete obstruction, and that this may be fatal or may be spontaneously relieved. He refers to a fatal case. Holt[11] believes that congenital syphilis may be an etiologic factor. As Cordes says: "The aforementioned causes certainly do not explain the greater number of the cases and other factors, probably developmental defects, coexist."

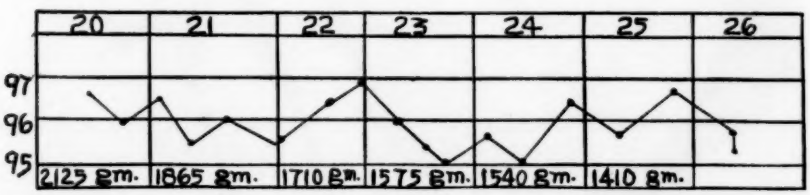

Fig. 1.—Temperature curve and weight variations.

Kreuter (quoted by Kaufmann[8]) asserts that there is absence or imperfect development of the lumen of the intestinal canal at a certain time in the development of the embryo (thirty to sixty days). At first the intestine is hollow, but as a result of hyperplasia of the lining epithelium, temporary closure results, which, in the normal fetus, later reopens. Failure to reopen gives various types of atresia or stenosis. Schridde denies this, having never found epithelial atresias. Forssner,[12] however, supports Kreuter's hypothesis, stating that just above the narrow point, as a result of swallowing amniotic fluid, there is enormous dilatation of that portion of the intestine with an accompanying hypertrophy of the muscularis. Purulent infiltration of the intestine and perforation may result. The lower segment collapses, the wall atrophies and the lumen may be totally obliterated.

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9. Drennan and Clark: Am. Jour. Dis. Child., 1915, **10,** 360.
10. Rowland, V. C.: Am. Jour. Dis. Child., 1915, **9,** 455.
11. Holt, L. E.: Diseases of Infancy and Childhood, 1916.
12. Forssner, H.: Hygiea, Stockholm, 1912, **74,** 1168; Ibid., Arch. f. klin. Chir., Berl., 1912, p. 477; Ibid., Zentralbl. f. Chir., Leipzig, 1913, **40,** 193.

Thorel's[13] case had seven points of atresia. He described the epithelial proliferation as the cause in his case and believed that it is the probable cause in most cases, unless there is rotation of the intestine, which, however, is very rare.

Quain[14] and Bailey and Miller[15] agree with Kreuter's theory as to the mode of origin of this condition, and considering the embryologic

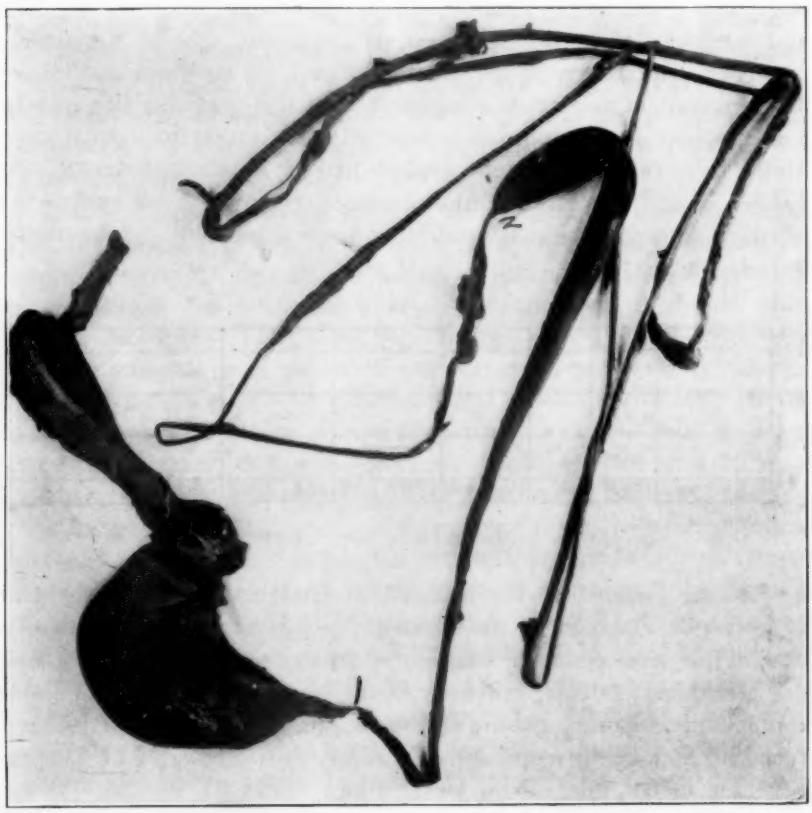

Fig. 2.—Large and small intestine showing strictures at 1 and 2

development of the intestine, it is evident that this process will account for a considerable number, if not the majority, of the cases of congenital intestinal obstruction.

The following symptoms, which were not recorded in our case, are of sufficient importance to remember in arriving at a diagnosis.

13. Thorel, F.: München. med. Wchnschr., 1899, **46,** 1202.
14. Quain: System of Anatomy, Embryology, **1,** Part 1, Ed. 10, 1896, p. 99.
15. Bailey and Miller: Textbook of Embryology, Ed. 1, 1910, p. 358.

Schwalbe[16] noted that in high stenosis the abdomen is retracted and in low stenosis the abdomen protrudes.

Pfaundler and Schlossmann[17] mention the following symptoms: Abdominal pain, obstinate constipation, deficient flatus, uncontrollable vomiting (at first food, later mucus, bile, blood and "kot"), meteorism, purposeless visible peristalsis, intestinal spasticity, tumor (Nothnagel's phenomenon) and finally collapse. They divide them into obstructions occurring in the small intestine and those in the colon and make a differential diagnosis according to the following table:

	Obstruction in Small Intestine	Obstruction in Colon (Lower Half)
Meteorism	Changeable, moderate and only epigastric lower and side parts of abdomen sunken.	Constant, strong and universal; "Kotwülste" palpable
Vomiting	None, or occasionally fecal	Fecal
Urine	Indicanuria, oliguria	No indicanuria; no oliguria
Collapse	Very severe	Moderate

REPORT OF CASE

The following case came to necropsy in Bellevue Hospital, Sept. 26, 1917.

History.—A girl, the second of twins, was born Sept. 20, 1917, of a healthy mother (Di R. M-3, service of Dr. J. C. Edgar, visiting obstetrician to Bellevue Hospital, to whom I am indebted for permission to abstract the clinical record). Family history negative; Wassermann on mother negative. At birth the patient was bleeding quite profusely from the nose and mouth. No other abnormality was noted. Shortly after birth the infant passed a small amount of meconium. The bleeding continued, although to a less extent, through the day, and at 8: 30 p. m., 10 c.c. of horse serum were given intramuscularly. Thereafter bleeding diminished very materially. The total amount of blood lost during this time was estimated at about 1½ ounces.

September 21. Immediately after the administration of a small amount of water the infant vomited a brownish fluid. When given the breast at 3 p. m., the child nursed exceedingly well, but a few minutes later vomited a thick brown residue. Blood oozed from nose and mouth throughout the day. At 5 p. m. 10 c.c. of the mother's blood was given, with but slight improvement in the child's bleeding. There was no bowel movement and the urine was scanty.

September 22. One ounce of saline was given into the rectum but was not retained; large amounts of mucus were expelled. Oozing of blood from mouth and nose occurred during the entire day, but stopped during the night. The child continued to vomit dark brown material after each feeding. There was no bowel movement.

September 23. Vomiting continued after each feeding. Vomitus this day was thick yellowish streaked with brown. Three ounces of olive oil given per rectum was not retained but was immediately expelled together with mucus. Bleeding continued. Pulse rapid (120), weak.

16. Schwalbe, E.: Morphologie der Missbildungen des Menschen und der Tiere. Ed. 1, 1909, **3**, 58.

17. Pfaundler and Schlossmann: Handb. der Kinderh., **1**, Part 1, Ed. 1, 1906, p. 72.

September 24. Eight drops of castor oil and saline enema given but without effect. Other symptoms as heretofore. Wrapped in cotton; kept warm.

September 25. Symptoms as before; bleeding continued; urine continued scanty.

September 26. Died at 1:55 p. m., following a profuse vomiting of yellowish material containing a large amount of blood.

Clinical Diagnosis.—Intestinal obstruction (congenital); hemorrhagic disease of the new-born.

Necropsy.—The body was that of an emaciated, white female infant, weighing 1,325 gm. and measuring 42 cm. in length. Rigor mortis was absent, and there

Fig. 3.—Microscopic section taken at 1 in Figure 2.

was a slight postmortem diffusion over the back. The entire skin was markedly icteric and there were ecchymotic areas over the lower extremities, on the back and the chest. The scalp was clean and covered by a fair amount of fine, long, black hair. Both fontanels were open and depressed. The pupils were equal and in middilatation; the sclerae were markedly icteric; conjunctivae free of petechiae and discharge. The nose and ears externally were negative. The lips were pale; the mucous membrane of the mouth was smooth. The tongue was dry, not fissured and there were no ulcerations present. The superficial lymph nodes were palpable only in the groin. The chest was symmetrical. The outlet of the thorax was acute. The abdomen was scaphoid. The external genitalia were normal. The extremities were normal.

The abdomen was free of fluid. The umbilical vein and both hypogastric arteries were patent. The greater portion of the large and small intestines was collapsed. There were numerous subperitoneal hemorrhages in the parietal and visceral peritoneum covering the intestines, the stomach and the liver. The mesenteric and retroperitoneal lymph nodes were not enlarged. The liver extended about 15 mm. below the costal margin. The diaphragm reached the fourth space on both sides. The stomach and about 9 cm. of the duodenum were distended with a dark semi-fluid material; and there was an apparently complete stricture of the duodenum, following which the jejunum, for a distance of about 20 cm., contained similar semi-fluid residue, and at this point there was another apparently complete stricture (Fig. 2). It was impossible to force the

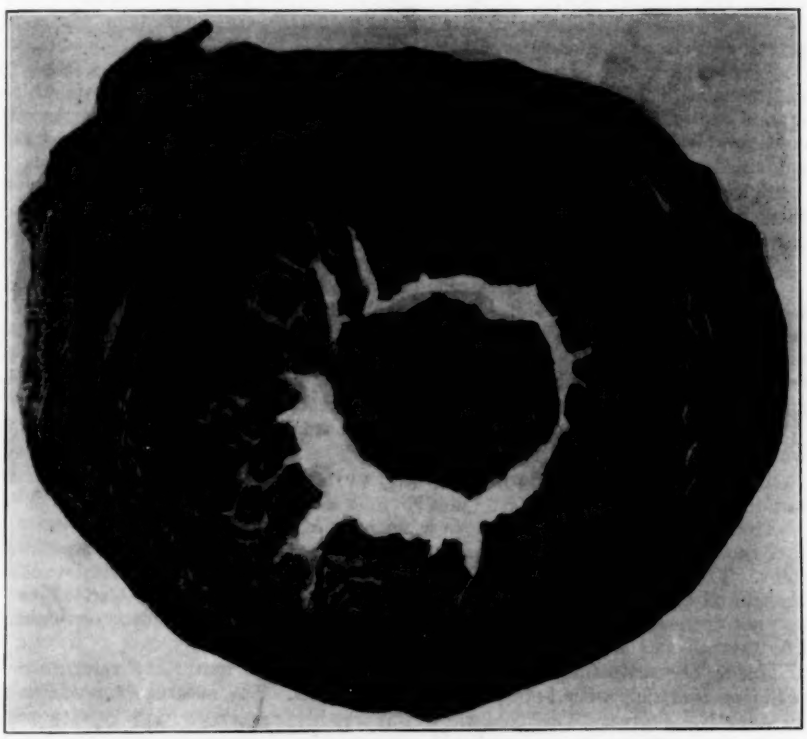

Fig. 4.—Microscopic section taken at 2 in Figure 2

intestinal contents through the stricture. The organs were in their proper position. There was no rotation of any portion of the intestine. The remainder of both the small and large intestines was totally collapsed. The appendix was small and lay free over the pelvic brim. A probe could be inserted into the rectum for a distance of 1¼ inches.

The right pleural cavity contained about 0.5 c.c. of a dark, hemorrhagic fluid. There were no adhesions. Over both the visceral and parietal pleurae of both lungs there were a number of large hemorrhages scattered in irregular fashion and presenting no definite demarcation. The pleural surfaces were smooth and of a bright red color, except where the irregular darkened hemorrhagic areas were found. The lung on section cut smoothly, presenting an irregularly reddish, mottled, crepitant surface, from which but little fluid could be expressed. The

bronchi were normal and were free of fluid. The peribronchial and mediastinal lymph nodes were not enlarged. The branches of the pulmonary artery were normal.

Heart: The precordial area was normal. The pericardial sac contained a small amount of dark hemorrhagic fluid and there were also some hemorrhagic areas on the epicardium. There were no petechiae present. The heart was of normal size, dark red in color. The epicardium was smooth. There were no subendocardial hemorrhages present. The heart chambers contained a moderate amount of dark red fluid blood. The valves were normal and apparently competent. The fossa ovalis was small, the foramen was patent. The myocardium was smooth and, on section, of a dark red color. The coronary vessels, as far as examined, were normal. The aorta and pulmonary artery were both smooth and elastic and without intimal change. The ductus arteriosus was closed.

The thymus was of normal size and weighed about 10 gm. On section it presented pale pink, glandular tissue.

Liver: This was of normal size. The capsule was not thickened. It was of a dark red color. On section it presented a firm, dark red surface from which a good deal of blood exuded. The vessels and duct were patent. The latter was considerably distended with bile. The gallbladder wall was smooth. The organ contained a small amount of thick, yellowish green bile.

Spleen: This organ was of normal size; it was firm. The capsule was not thickened. On section it presented a dark red surface in which the malpighian corpuscles were not prominent.

Pancreas: This was small, of normal consistency and the section was normal.

Kidneys: The kidneys were small and embedded in but little fat. They were considerably lobulated. The capsule stripped readily, leaving a pale reddish surface. On section there was distinct demarcation between cortex and medulla. The pyramids appeared a little swollen and contained well defined, radiating, orange-colored uric acid infarcts. The pelvis of the right kidney contained a few small, hemorrhagic areas. The ureters were normal.

The bladder was contracted and contained a small amount of urine. The wall was smooth and without lesion in the gross.

The adrenals were of normal size and on section, normal. There were no hemorrhages seen.

The uterus, tubes and ovaries were normal in the gross.

Organs of the neck and throat: The tonsils were small and pale pink in color. The trachea was normal in appearance. The esophagus to within about 1½ cm. of the cardia was normal. The remainder of the gastro-intestinal tract was not opened, but preserved in formaldehyd solution for subsequent sectioning. The thyroid was small; on section normal.

Head: The scalp was readily stripped from the calvarium. The anterior and posterior fontanels were both patent and depressed. The sutures overrode to a considerable degree. The calvarium was readily removed. The dura mater was normal. There was a considerable amount of pial edema present. The brain was soft. Section of the cerebral hemispheres was normal. The lateral ventricles were without fluid. There were no hemorrhages anywhere. The floor of the fourth ventricle had a decided yellowish tinge; otherwise it appeared normal. The examination of the middle ears and the sinuses accessory to the nose was negative.

Examination of the lower extremity of the right femur was negative.

Anatomic Diagnosis.—Congenital intestinal obstruction; hemorrhagic disease of the new-born (involving skin and serous surfaces); icterus neonatorum (obstructive jaundice?); uric acid infarction of kidney; pial edema, marked; patent umbilical vein, hypogastric arteries and foramen ovale.

Microscopic examination of the tissues and organs showed nothing remarkable.

Microscopic sections taken from the intestine at points indicated in the photographs (Figs. 3 and 4) show complete destruction of the lumen of the duodenum (1) and replacement by fibrous tissue in which there are a number of

blood vessels. There is no suggestion of a lumen. Section (2) made through the second point of constriction shows a lumen which is markedly diminished in caliber and occupied by a small blood clot.

The anatomic details of the foregoing case are in close agreement with those of three cases recorded by Ducros,[16] who found complete atresia of the duodenum-jejunum junction due to the presence of a fibrous mass replacing the lumen.

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